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Exploring the cognitive precursors of movement using a sensory-detection task

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Date: 7th August, 2008.

Abstract

Movement-related sensory suppression is an example of a motor-sensory interaction whereby sensory stimuli are perceived as less intense before and during a movement than at rest. This thesis focuses on sensory suppression of a weak electrocutaneous stimulus delivered just prior to movement onset, and develops this situation as a paradigm for studying motor-sensory interactions. In a typical experiment, subjects prepared a motor response in advance of a visual signal. They received a weak electric shock to the right hand just prior to voluntary movement. Subjects reported at the end of each trial whether they detected the shock or not. Movement-related sensory suppression was measured by comparing shock detection rates with non-movement control trials. This general paradigm was then used in a series of behavioural experiments which systematically manipulated cognitive processing prior to movement.

Premovement suppression was used to investigate the cognitive precursors of movement. First, it was shown that sensory suppression occurred for actions which were prepared, but then inhibited before execution. Second, sensory suppression was combined with the classic "stop-signal" paradigm. On trials where a stop signal came too late for subjects to inhibit their movement, a brief period of release from sensory suppression was nevertheless observed, as if the movement had been cancelled. This brief independence of sensory and motor systems suggests that the balance of excitation-inhibition is set separately for each system.

Third, when subjects performed pre-prepared sequences of movements, premovement sensory suppression was related only to the first movement in the sequence, and not to subsequent movements. This suggests that motor-sensory interactions are programmed at the level of individual movements, not sequences. Finally, delivery of an unexpected startling auditory stimulus produced an acceleration of sensory suppression corresponding to the well-known acceleration of motor reaction time. This finding suggests that subcortical motor circuits involved in startle may also contribute to sensory suppression. Overall, sensory suppression proved a useful tool for investigating the cognitive processes that take place prior to movement.

Acknowledgements

I would like to sincerely thank Prof. Patrick Haggard for his excellent and very stimulating supervision. I learnt a great deal and enjoyed working with the Motor Control Group.

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To Ma and Da,
Go raibh maith agat.

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CONTENTS

List of Figures

List of Tables

Chapter 1: Principles of motor-sensory interactions

The central nervous system (CNS) has a variety of mechanisms at its disposal that modulate the quantity and quality of the sensory information that it processes. One such mechanism is the suppression or "gating" of the transmission of cutaneous signals seen in association with voluntary movement. Studies in the rat, cat, monkey, and humans, examining somatosensory-evoked potentials (SEPs) (Brooke et al., 1997; Chapman et al., 1988; Coquery et al., 1972; Ghez and Lenzi, 1971; Rushton et al., 1981; Starr and Cohen, 1985) or single-unit responses (Chapin and Woodward 1982; Jiang et al. 1991), have demonstrated movement-related decreases in somatosensory transmission to the primary somatosensory cortex (SI) prior to and during movement. Movement-related gating also exerts powerful influences on perception. Thus, the detection of near-threshold cutaneous stimuli is decreased during movement (Coquery et al. 1971; Dyhre-Poulsen 1978), and detection threshold is increased accordingly (Chapman et al. 1987; Post et al. 1994). Psychophysical studies in humans have confirmed the existence of concomitant decreases in the detection of near-threshold stimuli prior to and during movement (Chapman et al. 1987; Pertovaara et al. 1992; Post et al. 1994). This thesis focuses on sensory suppression of a weak electrocutaneous stimulus delivered just prior to movement onset, as a paradigm for studying motor-sensory interactions.

This introduction starts with a description of a paradigm that provides an effective methodology for investigating movement-related sensory suppression, first demonstrated by Williams et al. (1998). This is the key paper for this thesis and a standard reference in the sensory suppression literature. Some of the factors that affect the sensory suppression of movement are then summarised. The neurophysiology of brain processes involved in movement and two mechanisms that can explain sensory suppression effects i.e. top-down and bottom-up inhibition, are discussed. Computational theories of motor control offer an alternative, though not necessarily exclusive, explanation of movement-related attenuation (e.g. Wolpert, 1997). According to this explanation, the central nervous system uses predictive mechanisms to attenuate self-produced stimuli relative to

externally-produced stimuli. Finally, an overview of the experiments contained in this thesis is given. It is proposed that premovement sensory suppression can provide an effective tool with which to explore the cognitive events that occur prior to movement.

1.1 A paradigm for sensory suppression

Williams et al., (1998), the key paper for this thesis, developed a simple, robust procedure for measuring the effects of sensory suppression. Their procedures have been replicated several times (Williams and Chapman, 2000; 2002; Chapman and Beauchamp, 2006). A brief description of the protocol and their main findings regarding the timing, magnitude and spatial extent of sensory suppression is now presented.

Williams et al. (1998) carried out a sensory detection task to measure the time-course of the detection of weak electrical stimuli. Subjects were trained to perform a rapid abduction of the right index finger (see Figure 1.1) in response to a visual signal. Shocks were set to be just detectable at rest (90% detection level).

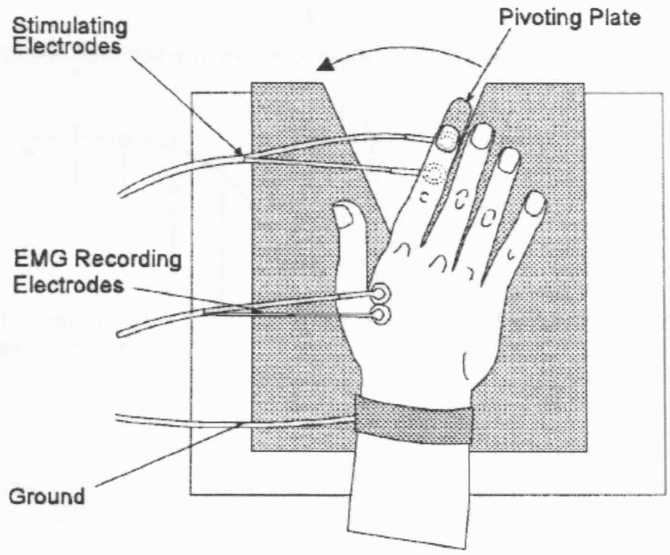

Figure 1.1. Experimental set-up from Williams et al. (1998) showing the pivoting plate on which the right finger rested (shown here in the initial rest position). Also shown are the positions of the stimulating electrodes (index finger) and the electromyographic (EMG) recording electrodes over the first dorsal interosseous (FDI) muscle.

Detection of shock stimuli delivered to the moving finger diminished significantly and in a time-dependent manner with the first significant decrease occurring 120 ms *before* the onset of EMG activity with detection rates describing a pronounced time-course (Figure 1.2). Williams et al. (1998) explained suppression prior to EMG onset in terms of top-down, descending inhibition i.e. inhibition induced directly by the outflow of motor commands onto afferent relay stations related to the preparation and execution of movement. In contrast, attenuation which occurred after EMG onset could be due to bottom-up masking of the shock stimulus by peripheral feedback generated from the movement.

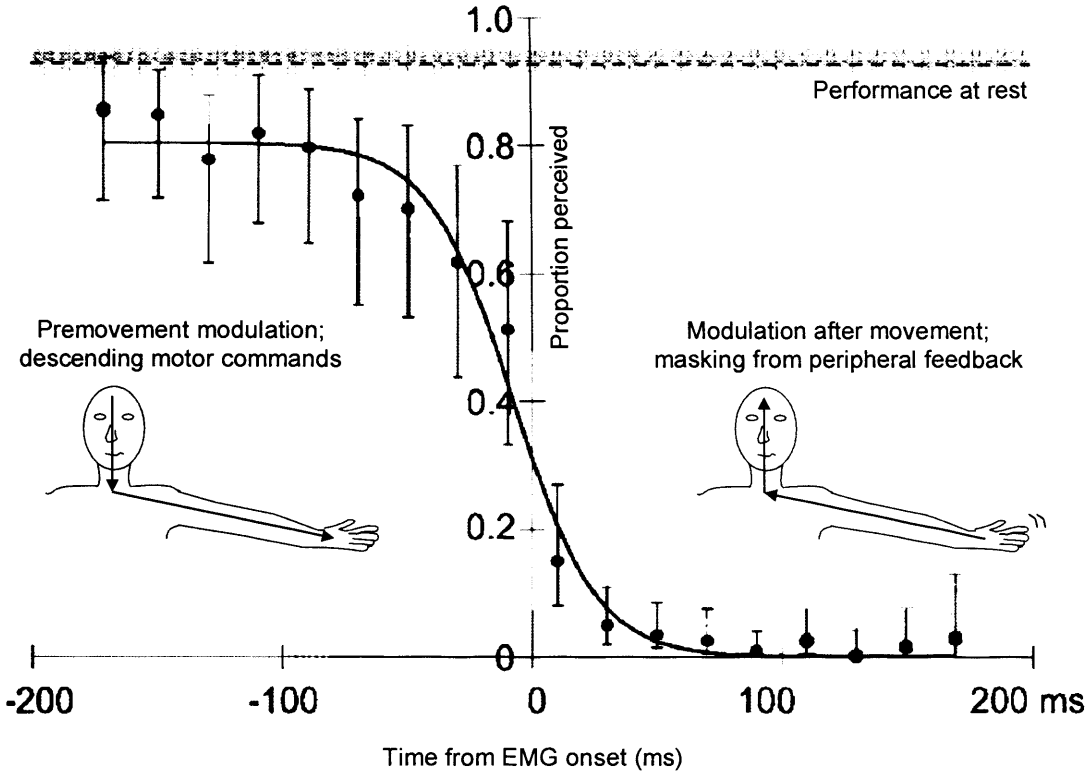

Figure 1.2. Effects of index finger movement on the detection of fixed intensity stimuli applied to the moving finger and performance at rest (Williams et al., 1998). Detection performance over time is plotted relative to the onset of electromyographic (EMG) activity (0 ms); a negative number indicates that the shock precedes EMG onset. Modulation that precedes the onset of movement and electromyographic (EMG) activity is interpreted as evidence that central signals relating to the preparation and execution of movement are contributing to sensory suppression. This premovement sensory suppression is of central interest for this thesis. Peripheral feedback generated during movement execution could also play an important role in the modulation that follows EMG and movement onset.

The procedure also allowed Williams et al. (1998) to demonstrate the importance of stimulus location in determining the timing and magnitude of the movement-related modulation of perception. They stimulated various parts of the body while the right index finger was moved. Reductions in detection were greatest and occurred earliest when the stimulus was delivered near the moving body part i.e. the right index finger. Time-dependent, movement-related decreases were also largely restricted to the moving digit, consistent with other studies (Coquery, 1978; Rushton et al., 1981). Time-dependent reductions in detection were smaller and occurred later as the distance between the

moving digit and the stimulation site was increased. When the shock was delivered to the passive left finger while the right finger moved, no sustained decrease in perceptual performance was observed. Therefore, sensory suppression is not just the result of the attentional demands of dual task performance. Williams et al. (1998) proposed that central mechanisms offered the best explanation of the results, consistent with previous findings of movement-related sensory suppression (Ghez and Lenzi, 1971; Jiang et al., 1990).

Movement-related modulation of the perception of weak electric shocks was dampened from approximately 90% detection rates 120 ms before EMG onset, to almost 0% at movement onset (Figure 1.2). Suppression that preceded the onset of electromyographic (EMG) activity and movement was interpreted as evidence that central signals related to the preparation and execution of the movement were at play. The effects of attenuation were also focused i.e. specific to the moving limb with little or no sensory suppression effects on more distant passive body parts. This basic effect has been replicated many times (Williams and Chapman, 2000; 2002; Chapman and Beauchamp, 2006; Pertovaara et al., 1994; Schmidt et al., 1990).

1.2 Factors influencing sensory suppression

Although there is wide agreement on the phenomenon of sensory suppression, there has been considerable variation between results with several factors affecting sensory suppression. Here we look at the generality of the sensory suppression effect across a range of different parameters and factors including spatial characteristics, stimulus intensity, movement parameters and the timing of sensory suppression effects.

1.2.1 What is the spatial extent of sensory suppression?

Insight into the mechanisms underlying movement-related decreases in transmission and perception comes from studies which define the spatial distribution of the gating influences. Several psychophysical studies have reported that proximity between the site of stimulation and the movement is an important factor with sites closer to the movement showing greater modulation (Coquery et al., 1971; Post et al., 1994; Schmidt et al., 1990; Rushton et al., 1981; Tapia et al., 1987). Attenuation of body SEPs (Rushton et al., 1981) and of percepts evoked by suprathreshold stimulation of the skin (Coquery, 1978) is maximal when the stimulated finger is moved. Movements of body parts remote to the point of stimulation do not gate transmission of the afferent input to the same extent in humans (Rushton et al., 1981; Pertovaara et al., 1994; Schmidt et al., 1990). In monkeys, on the other hand, the gating effects have been described as relatively widespread and non-specific (Jiang et al., 1990b).

1.2.2 Are there differences in sensory suppression between upper and lower limbs?

Sensory suppression may express itself quite differently in the upper and lower limbs. For upper limb somatosensory evoked potentials (SEPs), there is general agreement that the moved limb segment must be stimulated for effective movement-related gating to occur in cutaneous afferents (Rushton et al., 1981; Tapia et al., 1987). This localised specificity of sensory gating does not seem to apply for the proprioceptive afferents in the lower limbs of humans as, for example, movement of the contralateral leg also attenuates SEPs in the stationary leg (Staines et al., 1998). In contrast to the lower limbs which are mainly used for walking, separated control often characterises our upper limb movements. The exquisite control of the hand and especially the fingers in humans may require more sophisticated movement-related sensory feedback systems than do the lower limbs. Furthermore, tasks requiring digit movement probably have considerable associated reafference due to the high level of innervation in the digits. Movements about more proximal joints such as the elbows are likely to have less cutaneous feedback but

more proprioceptive feedback (Chapman, 1994). These factors may contribute to the differential sensory suppression that can be observed between the upper and the lower limbs.

1.2.3 Is sensory suppression affected by stimulus intensity?

Experiments using clearly suprathreshold stimuli and discrimination thresholds have shown minimal sensory suppression. For example, no decreases in perceived magnitude were observed when stimulus intensities ten times threshold were employed (Chapman et al., 1987). However, when intensity is limited to two times detection threshold, tactile detection is decreased but not abolished during finger movement, with the proportion of stimuli perceived during movement-related gating increasing as a function of stimulus intensity (Chapman et al., 1987; Milne et al., 1988; Post et al., 1994; Williams and Chapman, 2000). The time-course of the movement-related gating of detection remained invariant across a range of electrical shock stimulus intensities.

1.2.4 Do gating effects occur across different tactile sub-modalities?

Magnitude estimates of suprathreshold vibrotactile stimuli are diminished during movement (Chapman et al., 1987; Milne et al., 1988; Post et al., 1994; Dyhre-Poulsen, 1978). Sensations of flutter and pressure are also attenuated by movement though there is greater but equally graded gating of pressure than for flutter (Schmidt et al., 1990). These findings suggest that movement inhibits the tactile sensations of several tactile sub-modalities, thereby implying that the gating effects observed are generalisable to all cutaneous sub-modalities. However, this is not the case. Feine et al. (1990) reported no gating effects on the threshold for pain caused by heat during a motor activity task. This point is key to our understanding of sensory suppression. Once in the spinal cord, touch information proceeds upwards towards the brain via two major pathways. The spinothalamic pathway carries most of the information from thermoreceptors and

nociceptors and provides a mechanism for inhibiting pain perception. On the other hand, the dorsal-column-medial-lemniscal pathway conveys tactile and proprioceptive information that is used for planning and executing movement. Only the latter pathways show movement-related sensory suppression. The former pain pathways do show gating (Melzack and Wall, 1965), but not by movement. Thus, although the principle of top-down control of afferent input is very general, sensory suppression is a very specific form of motor-sensory interaction.

1.2.5 Movement parameters and sensory suppression

It has been demonstrated that passive movements can be as effective as active movements in generating movement-related suppression of tactile inputs (e.g. Chapman et al., 1988). A variety of mechanoreceptors are activated by passive movements, and evidence suggests that muscle spindle feedback contributes to the decrease in cortical SEPs during active and passive movement (Brooke et al., 1997). Though passive movements increase detection thresholds, the time-course of these modulations is radically different from those for active movement (Pertovaara et al., 1994). Chapman et al. (1988) found that the time-course for modulation of SEPS elicited by suprathreshold cutaneous stimuli was delayed considerably for passive as compared with active movements. Crucially, the modulation for passive movements followed movement onset instead of preceding it. This finding suggests that sensory suppression effects in passive movements can result from masking.

Presumably masking could also cause sensory suppression during active movements, but not before them. Movement-related gating is also a function of the kinematics of the movement, with faster movements producing larger suppression effects (Angel and Malenka, 1982; Rushton et al., 1981; Chapman et al., 1988; Rauch et al., 1985). Interestingly, movement per se is not essential for gating to occur. Cortical SEPs and shock detection thresholds are also modulated during the active phase of isometric muscle

contractions even though no actual movement of the effector ensues (Jiang et al., 1990; Williams and Chapman, 2002).

1.2.6 Timing and magnitude of movement-related sensory suppression

Knowledge of the timing of movement-related decreases in transmission and perception provides insight into the mechanisms underlying the gating influences. Modulation that precedes the onset of movement and electromyographic (EMG) activity generally is interpreted as evidence that central signals, related to the preparation and execution of the movement, play a role in the phenomenon (Chapman et al., 1988; Coulter, 1974; Dyhre-Poulsen, 1978; Ghez and Lenzi, 1971; Jiang et al., 1990). Peripheral feedback, generated during movement execution, also plays an important role in the modulation that follows movement onset (e.g., Chapman et al., 1988; Huttunen and Hömberg, 1991; Jones et al., 1988). Wide variations have been reported with regards the timing of the onset of movement-related gating. Studies describe suppression ranging from 400 ms (Seki et al., 2003) to 200 ms before the onset of movement (Chapman et al., 1988; Ghez and Lenzi, 1971; Coulter, 1974) to 30 ms after movement onset (Kristeva-Feige et al., 1996). There is also considerable variation in the magnitude of the gating effects reported, ranging from 7.5% (Coulter, 1974) to more than 90% reduction in detection rates (Kristeva-Feige et al., 1996). The differing effects reported in the literature can probably be partly explained by differences in protocol design including factors such as stimulus intensity.

1.2.7 Summary and discussion; factors influencing sensory suppression

Sensory suppression is a robust example of an interaction between the sensory and motor systems and is found generally across different sensory inputs, movements, methods, and dependent variables. Attenuation of percepts evoked by stimulation of the skin is maximal when the stimulated hand moves, while movements of body parts remote to the point of stimulation do not gate transmission of the afferent input to the same extent.

However, this localised specificity of sensory gating may not apply to the lower limbs in humans. Time-dependent sensory suppression occurs for stimulus intensities ranging from threshold to approximately two times threshold. Gating effects are generalisable to many cutaneous modalities including electrical, vibrotactile, pressure and flutter. However, not all inputs are gated e.g. there is no gating of pain caused by heat while performing a motor task. Passive movement can be as effective as active movements in generating movement-related suppression of tactile inputs. Movement-related gating is also a function of the kinematics of the movement, with faster movements producing larger gating effects. However, the time-course of these modulations is radically different with modulation for active movement preceding movement onset. Knowledge of the timing of movement-related decreases in transmission and perception provides insight into the source of the gating influences. Modulation that occurs after EMG and movement onset results from masking caused by peripheral feedback. Alternatively, and of special interest for this thesis, modulation that precedes the onset of movement and electromyographic (EMG) activity is interpreted as evidence that central signals related to the descending motor commands are evoked.

The gating of active movement presents us with a paradox; sensation is reduced or gated during active movement; yet when we explore an object or fabric with our fingertips one would hardly think that gating occurs. It seems strange that sensation is suppressed when it could be most useful. The advantage that active touch enjoys is that the collection of sensory impressions can be controlled. The exploring digits can be manoeuvred so that the most sensitive part of the skin comes into contact with the relevant surface, and movement speeds can be accelerated or braked at critical times during exploration to reduce gating effects (Lederman and Klatzky, 1987). Slow searching movements require much more feedback and hence are associated with less gating of cutaneous input (Schmidt et al., 1990). Thus, motor strategy during exploratory active touch can be used to override any disadvantage of gating during movement. Additionally, central processes such as attention and motor set might enhance the transmission of relevant inputs.

1.3 Neurophysiology of brain processes involved in movement

The cortex is involved in initiating our behaviour and in executing all different kinds of movements, yet all of these movements are brought about with the cooperation of the subcortical structures including the basal ganglia and the cerebellum. These structures handle the information from and to the cortex via different pathways. Both have connections to structures in the brainstem from which spinal motoneurons are activated. Two loops operate sequentially (Goldberg, 1985). In the first (medial) loop, virtually all regions of the cerebral cortex project via the basal ganglia and motor nuclei of the thalamus (e.g. Strick et al., 1995) back to regions of the cortex, including the supplementary motor area (SMA). In the second (lateral) loop, activity from somatosensory and posterior association areas is projected back through the cerebellum and specific motor nuclei of the thalamus to the primary motor cortex (Allen and Tsukahara, 1974). The function of this feedback-dependent loop is thought to be the context-dependent adjustment of the parameters of the first loop by translating sensory information into immediate adjustments of motor activity to improve the timing and smoothness of actions (Goldberg, 1985; Liu et al., 1999).

The motor parts of the cortex include motor cortex (M1), the premotor cortex (PMC) and the supplementary motor area (SMA) and pre-SMA (Figure 1.3). The motor cortex is involved in the execution of voluntary movement. The major output from the motor cortex to the spinal cord and muscles is formed by the pyramidal tract. Movement starts with the discharge of pyramidal tract neurons (Evarts, 1968). Axons of the pyramidal cells descend to the brainstem, premotor cortex (PMC) and spinal motoneurons either directly or via interneurons. Discrete skilled movements are mediated via this pathway. Preparation and initialisation of movements seem to be organised in the pre-SMA and the SMA (Wiesendanger, 1993). Voluntary movements are preceded by the Bereitschaftpotential or readiness potential, an electrophysiological response measured using scalp electrodes, which starts over the M1-SMA complex, approximately 1.5 seconds before overt movement (Kornhuber and Deecke, 1965). The SMA-proper, is thought to be involved in simple or overlearned tasks whereas the pre-SMA, lying just

rostral to the SMA proper, is thought to be more involved in complex tasks (Carbonnell et al., 2004).

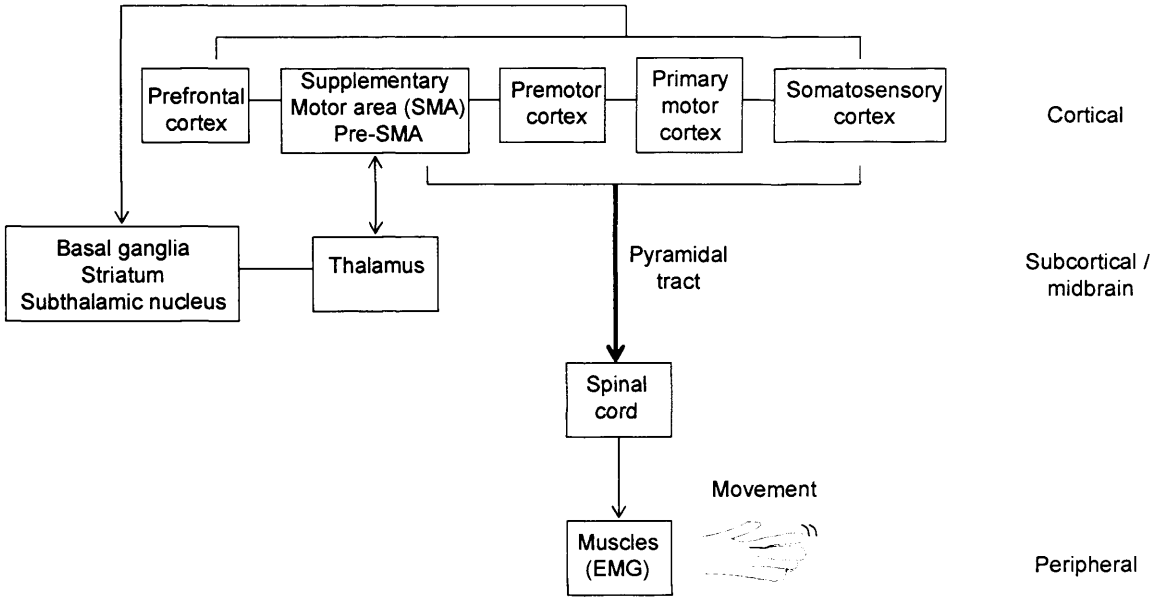

Figure 1.3. Schematised diagram (based on Band and van Boxtel, 1999) containing some of the main anatomical structures in the brain involved in movement.

1.4 Neural mechanisms of sensory suppression

Two main mechanisms have been proposed to explain movement-related gating of cutaneous input, a top-down and a bottom-up mechanism. These two mechanisms can exert different modulating effects on the perception of tactile stimuli.

1.4.1 Top-down sensory suppression

The concept of descending inhibition i.e. inhibition induced directly by central motor commands descending on afferent relay stations, is supported by the fact that that sensory suppression may precede movement (Ghez and Pisa, 1972; Coquery, 1978; Milne et al., 1988). The amplitude of somatosensory-evoked potentials (SEPs) is also decreased prior

to the onset of movement and movement-related electromyographic (EMG) activity (Chapman et al., 1988; Cohen and Starr, 1987; Coulter, 1974; Ghez and Lenzi, 1971), i.e., before the generation of peripheral feedback from the muscle. Descending motor inhibition is central to this thesis.

1.4.2 Bottom-up sensory suppression (Masking)

Peripheral feedback generated by movement is also considered to be an important source of gating signals by masking. A tactile stimulus (e.g. a shock) can lead to a percept that is reportable by subjects and / or a cortical response which may be recorded from electrodes attached to the scalp (somatosensory evoked potential). However, background noise generated by movement can be of greater magnitude than the signal triggered by the tactile stimulus. Hence, the background noise can interfere with the processing of the shock thereby attenuating its percept by altering the signal to noise ratio (Blumenthal et al., 2006). The principal evidence for ascending inhibition comes from several studies that have demonstrated that passive movements can also diminish the amplitude of SEPs (Brooke et al., 1997; Huttunen and Homberg, 1991; Rushton et al., 1981; Staines et al., 1998) but with a time-course that is different from that seen for active movements because the modulation occurs only after movement onset (Chapman et al., 1988).

1.4.3 Neural loci of sensory suppression signals

There are several sites where sensory suppression could occur; either in the cortex or blocking access of afferent ascending transmission to the cortex. Thus, there are several possible sites of motor-sensory interaction. A key question is whether these sources occur in motor preparation areas upstream of the motor command generation in M1, or in areas associated with motor execution such as M1 itself. Cortical stages of the motor hierarchy which prepare motor commands contribute to the production of sensory suppression of voluntary movements suggesting that suppression arises from premotor

command signals (Voss et al., 2006; Figure 1.4). A similar conclusion was reached by Haggard and Whitford (2004). Both primary motor cortex and somatosensory cortex (S1) are also thought to be important sources of central modulatory influences (Jiang et al., 1990; Chapman, 1994; Chapman and Beauchamp, 2006) via their descending projections to the various relay sites in the dorsal-column-medial-lemniscal pathway. Evidence for a central role in sensory attenuation for primary motor cortex (area 4) has been demonstrated by Jiang et al. (1990b) who showed that discrete activation of motor cortex using sub-threshold intracortical microstimulation (ICMS) can diminish the amplitude of S1 cortical SEPs in monkeys, possibly via collaterals from the pyramidal tract to the dorsal-column nuclei.

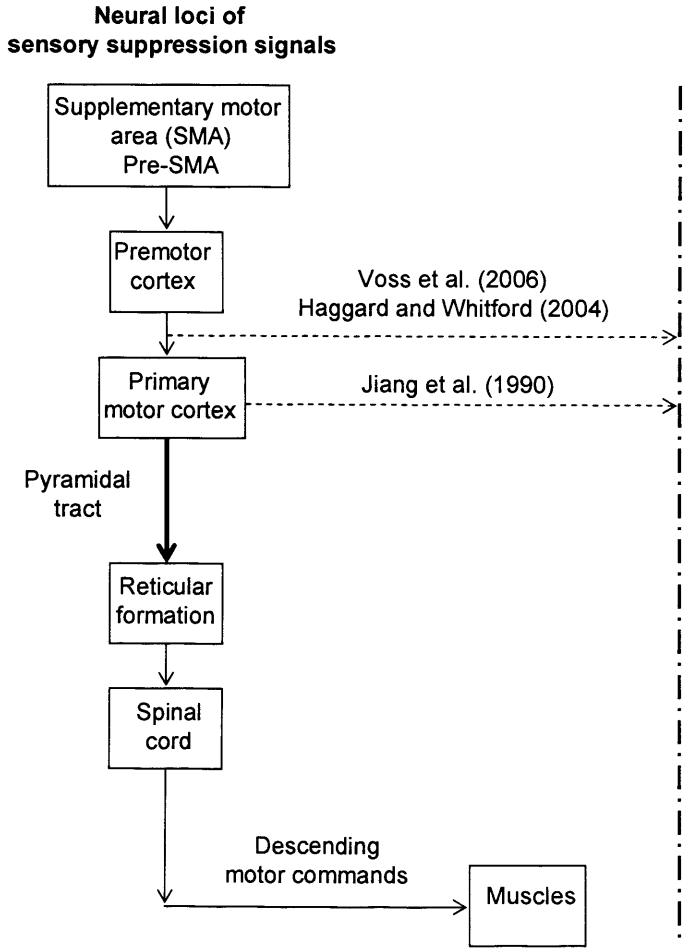

Figure 1.4. The efferent pathway. Motor information from the cortex is transmitted to the periphery via the pyramidal tract. Sensory suppression has been shown to originate from premotor command signals and from primary motor cortex (dashed arrows).

1.4.4 Destinations of sensory suppression

During normal voluntary movement, reafferent feedback from the periphery continuously converges on the spinal circuits that are activated by descending motor commands. These cutaneous inputs are presynaptically-inhibited at the level of the spinal cord during wrist flexions in monkeys (see Figure 1.5; Seki et al., 2003). Additionally, cutaneous afferent signals are relayed through the two dorsal-column nuclei (DCN) and the sensory thalamus on the pathway from the periphery up to somatosensory cortex (S1). Sensory attenuation may potentially be exerted at the dorsal-column nuclei (e.g. Gordon and Jukes, 1964) and S1 (e.g. Rushton et al., 1981; Chapin and Woodward, 1982). The suppression seen at the dorsal-column nuclei, or at the spinal cord, can precede the onset of EMG activity in the active limb and so is generally considered to be largely central in origin (Chapman et al., 1988; Coulter, 1974; Ghez and Lenzi, 1971). Also, responsiveness to cutaneous stimuli in the primate thalamus is diminished prior to and during active movement (Chapman et al., 1988).

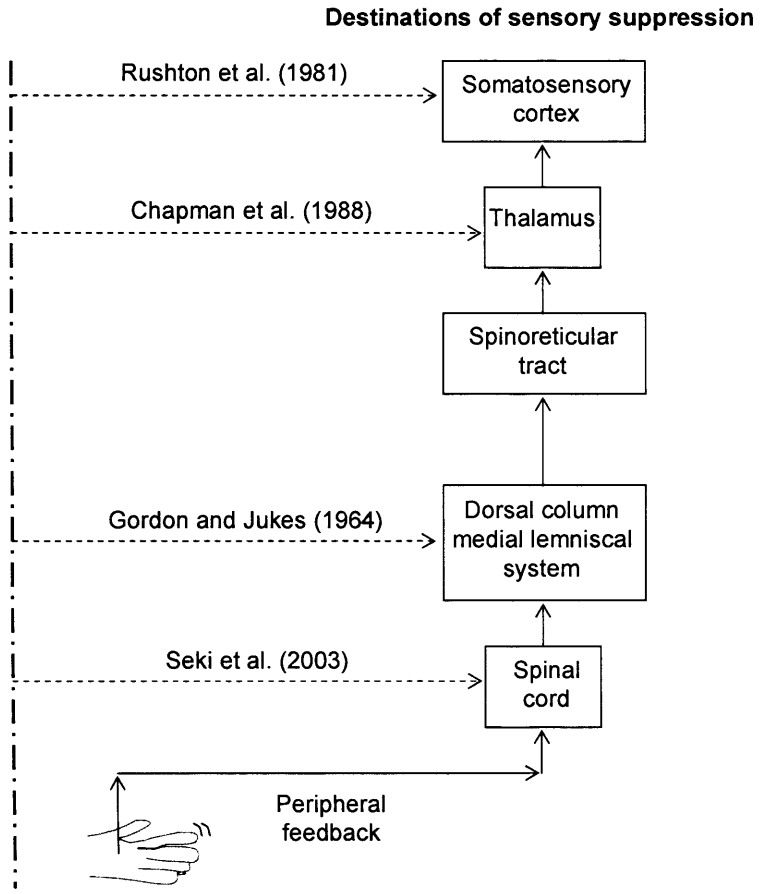

Figure 1.5. The afferent pathway. Sensory information from the limbs (i.e. non-painful tactile sensation and proprioception) is transmitted to the thalamus by the dorsal-column-medial-lemniscal pathway. Thalamic neurons then send their axons to primary somatosensory cortex. The dashed arrows refer to sites where sensory suppression effects have been measured at several levels of the afferent pathway.

1.4.5 Summary; neural mechanisms of sensory suppression

Overall, sensory suppression appears to be a highly general form of motor to sensory interaction rather than a single specific physiological circuit (Figure 1.6). Multiple structures probably contribute to movement-related gating, and the relative contribution of each appears to vary across the chain of events that ultimately leads to perception (Schmidt et al., 1990).

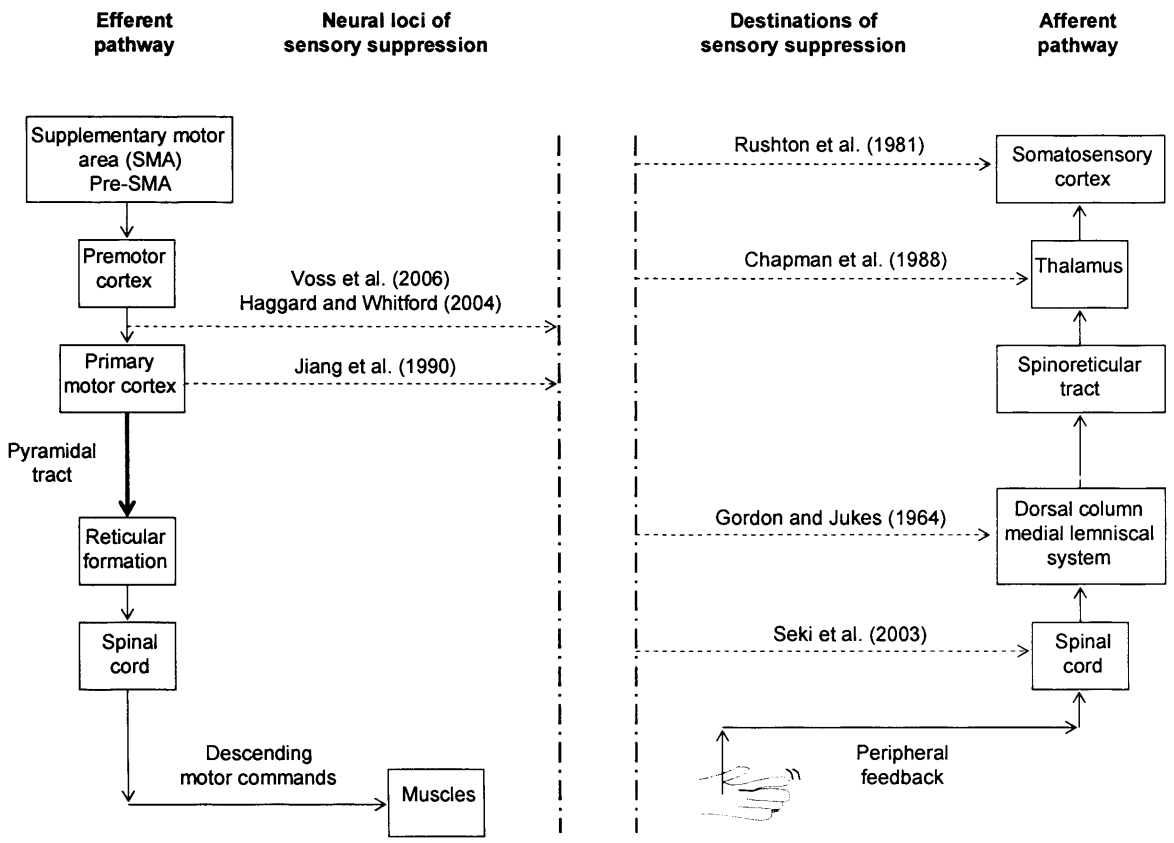

Figure 1.6. Schematic of afferent (locations where attenuation occurs) and efferent (signals causing sensory suppression) pathways showing some representative possible motor-sensory interactions (horizontal dashed arrows).

1.5 Computational significance of sensory suppression

1.5.1 Computational motor control theories

The motor system can be considered a control system in which the input is the motor command that produces a movement and the output is the sensory consequence of that movement (Wolpert, 1997). On the basis of the current state of the system, the desired goal and a model of the system itself, the central nervous system (CNS) can compute a sequence of motor commands that should generate the movement required to reach the goal. This is called the inverse model (see Figure 1.7).

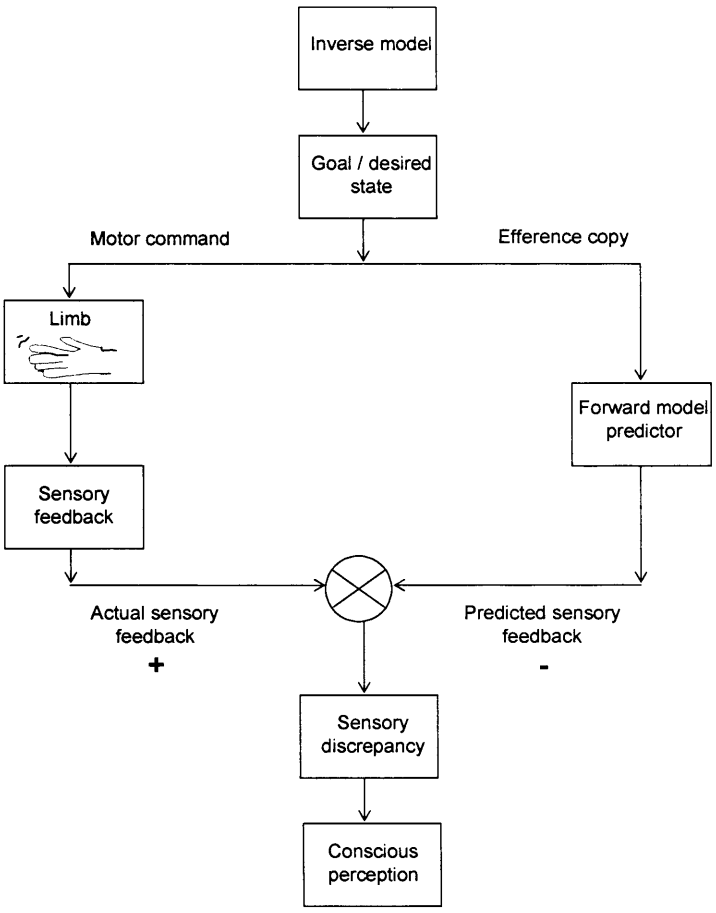

Figure 1.7. Computational model of motor control (after Blakemore et al., 2002). An internal forward model makes predictions of the sensory feedback based on an efference copy of the motor command. These predictions are then compared to the actual sensory feedback to produce the sensory discrepancy. Conscious perception registers sensory discrepancy rather than afferent information.

Using a predictive forward model, the CNS can also use the outflow of motor information to generate a model of the sensory inflow before the inflow has actually occurred. Forward models are therefore useful at explaining fundamental problems in motor control. As the delays in most sensorimotor systems can be large, feedback control would be too slow to achieve rapid movements. With the use of a forward model, the outcome of an action can be estimated and used before feedback even becomes available.

1.5.2 Sensory suppression and the theory of computational motor control

In order to avoid responding to sensory inflow that arises from self-generated actions, the sensory system needs to know what the motor system has done. Self-monitoring, computational mechanisms have previously been the subject of much investigation, originally in the oculomotor domain (e.g. von Holst and Mittelstaedt, 1950; Sperry, 1950). However, it appears that sensory predictions produced in conjunction with motor commands are not restricted to eye movements but also provide perceptual stability in the context of many other self-produced actions. Our ability to monitor, and recognise as our own, self-generated limb movements, touch, speech, and thoughts suggests the existence of a more general mechanism (Frith 1992). Wolpert and colleagues (Bays et al., 2005; 2006; Blakemore et al., 1999; Shergill et al., 2003) proposed that the motor command, via an efference copy (Figure 1.7), gates the perception of the intensity of self-induced tactile sensations. The forward model makes predictions of the sensory feedback based on the motor commands. These predictions are then compared to the actual sensory feedback to produce the sensory prediction errors or sensory discrepancy. When the actual and the predicted sensory feedback are of equal magnitude they cancel, and less sensory information is processed. The lower the sensory discrepancy, the greater is the attenuation of tactile sensation. The prediction can thus be used to anticipate and compensate for the sensory effects of movement; attenuating the component that is due to self-movement from that due to changes in the outside world.

Evidence that a self-produced tactile stimulus is indeed less intense than an externally generated stimulus was provided by Blakemore et al. (1999) who used functional neuroimaging (fMRI) to examine neural responses when subjects experienced a tactile stimulus that was either self-produced or externally-produced. Subjects either tickled themselves on the left palm or were tickled by the experimenter via a robotic interface (Figure 1.8).

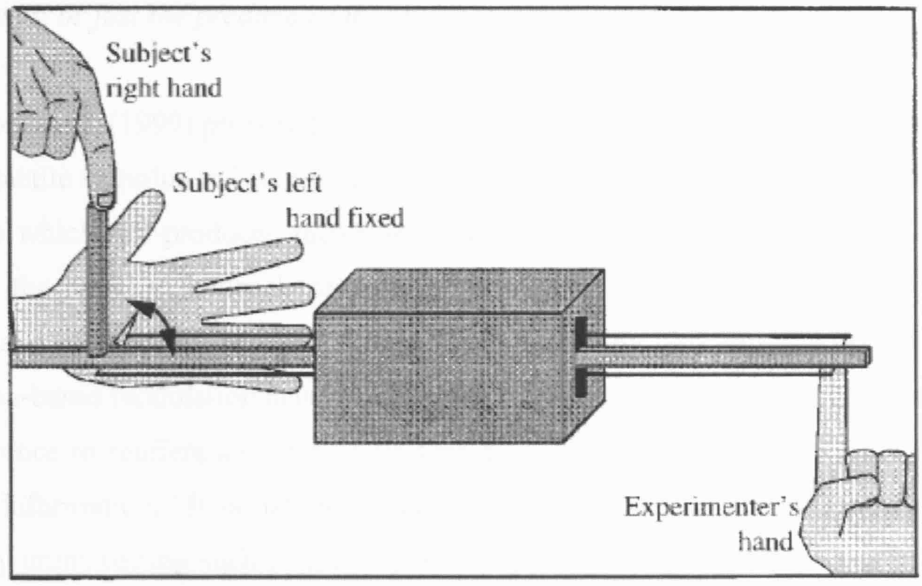

Figure 1.8. Diagram of experimental set-up used by Blakemore et al. (1999). A tactile stimulus device consisted of a piece of soft foam attached to a rod, which could pivot about its centre. The foam made light contact with the subject's left palm. The rod could be moved either by the subject using the right hand or by the experimenter.

Self-generated tickle stimulation was perceived as less ticklish than the same tickling stimulation that arose when the stimulation was externally-produced. When an artificial delay or trajectory perturbation was introduced between a movement and the resultant tactile stimulation, then self-generated stimulation was also rated as more ticklish. As the discrepancy between predicted and actual sensory feedback was increased during self-produced tactile stimulation, there was a corresponding decrease in the level of sensory attenuation and an increase in tickliness. Furthermore, neuroimaging data showed that more activation was found in somatosensory cortex when the stimulus was externally-produced than when self-produced, consistent with previous reports of sensory gating (e.g. Rushton et al., 1981).

1.5.3 Are all or just the predicted consequences suppressed?

Blakemore et al. (1999) proposed that a necessary requirement of predictive attenuation is that the tactile stimulus and its causal motor command correspond in space and time. The extent to which self-produced tactile sensation is attenuated is proportional to the error between the sensory feedback predicted by the internal forward model of the motor system and the actual sensory feedback produced by the movement. Therefore, prediction-based modulation acts as a filter for incoming sensory signals that can enhance the afference to reafference ratio. Our sensory systems are constantly bombarded with sensory information. It is therefore important to filter out sensory information that is relatively uninteresting such as the sensations of our own movements, so as to be better able to pick out sensory information that carries evolutionary importance e.g. someone else touching us or a wasp landing on our arm. These results suggest that the perceptual attenuation of self-produced tactile stimulation is due to a precise modulation of the sensory feedback, based on specific sensory predictions, rather than a non-specific movement-related attenuation of all sensory signals.

1.5.4 Predictive sensory attenuation is event-driven

Bays et al. (2005) provided further evidence for such specificity and proposed that predictive tactile sensory attenuation is event-driven i.e. it is linked to specific external events arising from movement rather than to the actual movement per se. In their task, subjects used their right index finger to tap a force sensor mounted above their left index finger (Figure 1.9).

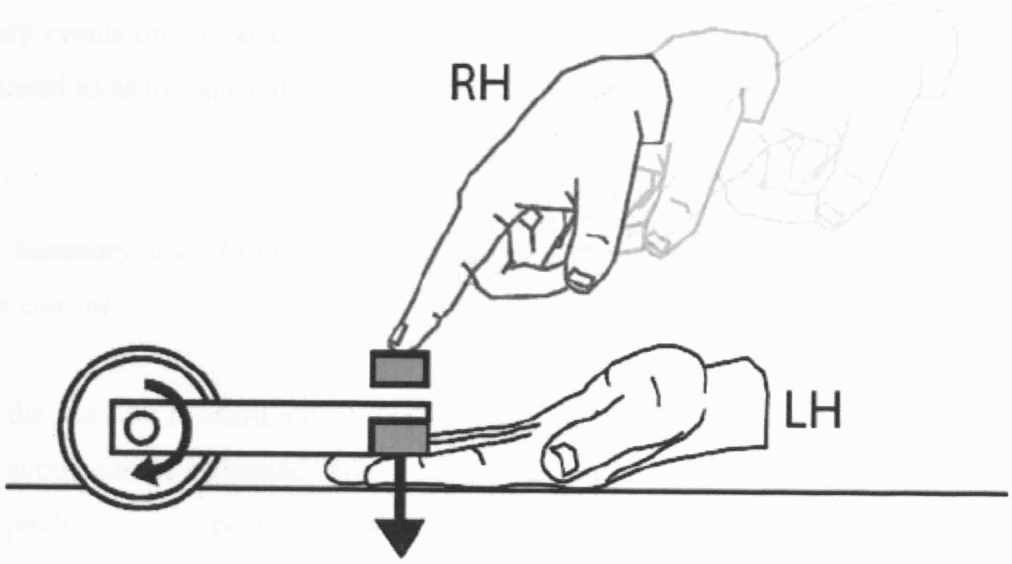

Figure 1.9. Apparatus used by Bays et al. (2005). Subjects made a speeded movement to produce a brief force pulse (active tap) with their right index finger on a second force sensor fixed above their left index finger. A similar force pulse (test tap) was delivered to the left index finger by a torque motor.

Subjects were required to judge the relative magnitude of two taps experienced sequentially on the left index finger. When a motor generated a tap on the left finger synchronously with the right tap, simulating contact between the two fingers, the perception of force in the left finger was attenuated compared to the same tap experienced during rest. However, no attenuation was observed when left taps were triggered by right finger movements that stopped short of or passed wide of the sensor. The event of one hand contacting the other, even via the indirect mechanical linkage, was required for attenuation. These results provide evidence for precise predictive sensory attenuation that does not result from either movement or sensation in the active effector alone, but rather is linked to task-specific events predicted to arise as the consequence of an action. Moreover, when one finger made a tapping movement above the finger of the other hand, sensation in the passive finger was attenuated only when contact was expected between the fingers (Bays et al., 2006). The level of attenuation when contact was expected was the same regardless of whether actual contact occurred or not. These results are

consistent with the action of a predictive online process that can predict self-generated sensory events on the basis of the planned motor activity. Incoming sensory signals are attenuated so as to reduce the sensory salience of those predictable events.

1.5.5 Summary and discussion; sensory suppression and the theory of computational motor control

With the use of a forward model, the CNS can mimic sensory feedback and the outcome of an action can be estimated and used before actual sensory feedback becomes available. This prediction can be used to anticipate and compensate for the sensory effects of movement; attenuating the component that is due to self-movement from that due to changes in the outside world. Thus, prediction-based modulation can act as a filter for incoming sensory signals. If the sensory consequences of a movement are predicted accurately, they need not be perceived – to do so would clutter consciousness superfluously. Evidence for this predictive mechanism has come from studies where self-generated tickle stimulation was perceived as less ticklish than the same tickling stimulation that arose when the stimulation was externally-produced. Predictive tactile sensory attenuation seems to be event-driven i.e. it is linked to specific external events arising from movement rather than to the actual movement itself.

There are differences and similarities between the mechanisms proposed for attenuation by Williams et al. (1998) and Wolpert and colleagues (e.g. Bays et al., 2005; 2006; Blakemore et al., 1999; Shergill et al., 2003). One difference is that Williams et al. (1998) use an *externally*-imposed stimulus (an electric shock delivered by a device) whereas Wolpert and colleagues' paradigm typically involves *self-generated* touch to the passive hand with the active hand via a mechanical interface. A common finding between both paradigms is the notion that the motor command contributes to suppressing sensory inputs prior to and during movement. However, in the case of Williams et al. (1998), a role for the central motor command was only obtained when the moving hand was stimulated i.e. premovement attenuation was specific to the moving hand with little

or no attenuation on the contralateral hand. In contrast, the suppression of self-induced sensations reported by Wolpert and colleagues is elicited by contralateral movements (e.g. the moving left hand elicits attenuation on the contralateral passive hand; see Figures 1.8 and 1.9). This evidence suggests that an internal forward model is able to provide information about the motor commands sent to one hand, which is used to cancel the resultant tactile sensation in the other hand. Williams et al. (1998) found no evidence for a time-dependent decrease in tactile perception of externally-imposed stimuli during contralateral movements. Under certain circumstances, self-induced and externally-imposed tactile sensations may be differentially controlled. For example, Bays et al. (2005; 2006) found that self-induced inputs are suppressed only when the subject *expects* to generate sensory input. Actual movement was not necessary for attenuation. This implies a predictive mechanism that serves to enhance perception of externally-generated inputs. The brain has a number of mechanisms at its disposal that operate at a number of levels within the motor hierarchy. Depending on the context, one or more of these mechanisms may be deployed to ensure behaviour that is appropriate to the specific situation. Though the evidence suggests that there are some fundamental differences in the underlying mechanisms between the predictive mechanism of Wolpert and colleagues and that proposed by Williams et al. (1998), the functional role of movement-related suppression i.e., to enhance salient stimuli, may be the same for both accounts.

1.6 Sensory suppression and the cognitive precursors of movement

All motor control models assume that making a voluntary movement involves a number of processes or stages that precede movement (Sternberg, 1968; Rosenbaum, 1983; Wolpert, 1997; Crammond and Kalaska, 2000). Two such paradigms used to investigate motor preparation, execution and inhibition processes are the Go/NoGo task (e.g. Hester et al., 2004; Hoshiyama et al., 1996; Kalaska and Crammond, 2005; Rubia et al., 2001) and the stop-signal paradigm (for a review, see Logan, 1994). In the Go/NoGo task, subjects respond to a Go signal and attempt to withhold a response when a rare NoGo signal occurs. In the stop-signal paradigm, the Go signal is occasionally followed by a

stop signal *within* a trial. Thus, subjects must attempt to stop a response process that is presumably already underway. The main contrast between the two paradigms can be expressed in terms of competition between alternative motor plans. In Go/NoGo paradigms, there is no explicit competition between movement plans. On a single trial, a Go signal triggers a serial process that results in overt movement *or* a NoGo signal results in the cancellation of the prepared movement. The task is all-or-nothing, either to go or not to go. In stop-signal paradigms, the movement plans cued by the Go *and* the subsequent presentation of a stop signal within the same trial, are placed in *direct competition* (Figure 1.10).

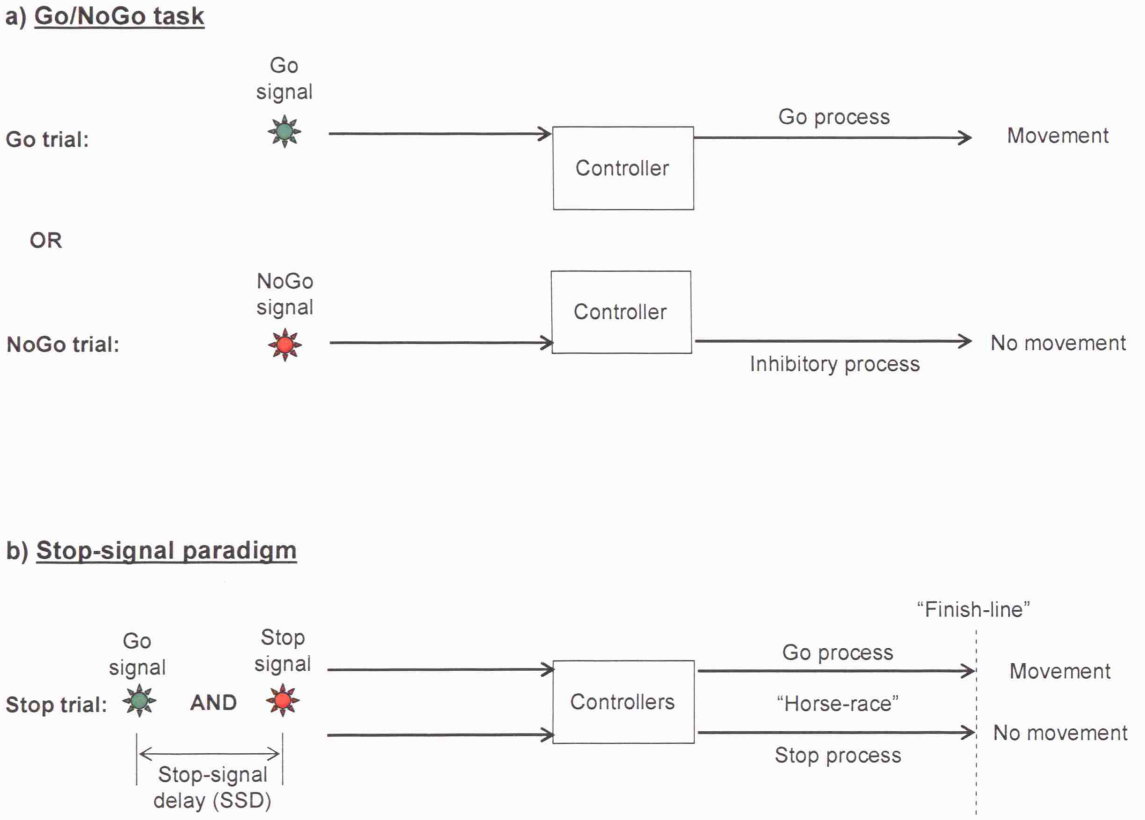

Figure 1.10. Schematic of Go, NoGo and stop trials in the Go/NoGo and stop-signal paradigms. A top-down controller shunts the commands generated by the visual signals into the appropriate response. a) During the Go/NoGo task, the response is either to go *or* not to go. b) Stop-signal paradigm. On a stop trial, the stop signal is presented at a variable time delay (called the "stop-signal delay" or SSD) after the Go signal. Thus, the activation of the go process *competes* directly against the activation of the stop process in a "horse-race" towards a winner-takes-all threshold "finish-line" that determines whether a movement is executed or not.

The level of competition is critically biased by the delay between the presentation of the Go signal and the stop signal. The two competing processes are envisaged as racing against each other independently, with the outcome being a monotonic function of the speed of the stop and the go processes and the delay between them (known as the "stop-signal delay" or SSD). Competition between the go and stop processes is maximised by adjusting the SSD during the course of the experiment as performance varies, so that a successful change of plan occurs on approximately half of stop trials.

Psychologists have classically studied sensory suppression in relation to movement (Brooke et al., 1997; Chapin and Woodward 1982; Chapman et al., 1988; Coquery et al., 1972; Ghez and Lenzi, 1971; Rushton et al., 1981; Starr and Cohen, 1985). However, the classic Go/NoGo and stop-signal paradigms emphasise the essential role that *inhibition* plays in the control of movement. An important aim of this thesis is to use these paradigms to study the sensory consequences of movements that are prepared, but then cancelled.

1.7 Overview of experiments

Chapter 2 will describe an experiment that attempted to replicate the main findings of Williams et al. (1998) to confirm that sensory suppression can occur in advance of physical movement. This methodology was then used to probe the motor-sensory interactions that precede movement. An important aspect of behavioural control is the inhibition of movement. It is not known how sensory suppression unfolds when a prepared movement is cancelled. Experiments exploring this question will be described in Chapters 3, 4 and 5. Whether an action is executed or withheld can ultimately depend upon a delicate balance of excitatory and inhibitory processes. How the brain processes sensory information when motor preparation is unpredictably switched on and off is investigated in Chapters 6 and 7. In some situations, one hand is primed for movement but then unexpectedly the other hand is recruited. Chapter 8 will describe an experiment that explored such situations by analyzing premovement sensory suppression effects

during a "Posner-type" action paradigm. Next, when subjects prepare a sequence of movements, the sequence representation is thought to be stored as a chunk in advance of the movement (Lashley, 1951). However, it is not known whether sensory suppression is affected by all movements in the sequence or just by the first element. Sensory processing prior to carrying out a pre-prepared sequence of movements is evaluated in Chapter 9. Finally, Chapter 10 investigates if sensory suppression remains intact in advance of a movement even when the movement is rapidly accelerated by delivering an unexpected startling auditory stimulus. To close, the main findings of the current series of experiments and some plans for future experiments are then discussed.

Chapter 2: Suppression of sensory detection prior to movement; a behavioural paradigm and a replication

2.1. Abstract

The purpose of the first experiment was to replicate the main findings of the study by Williams et al. (1998) in relation to the time-course, magnitude and spatial extent of sensory suppression effects. This is the key paper for this thesis and a standard reference in the sensory suppression literature. Subjects performed a motor task (movement of the index finger) and a perceptual task (detection of a weak electrical shock delivered to the right index finger). Subjects were instructed at the start of each trial if they should move or not. Results showed that shock detection rates on movement trials were less than on non-movement trials replicating the sensory suppression effects demonstrated by Williams et al. (1998). Furthermore, and of central interest for this thesis, detection rates for shocks delivered in the interval just prior to electromyographic (EMG) onset were also substantially reduced. Sensory suppression was focused on the moving digit in line with the original study. We conclude that premovement sensory suppression can be used as a tool to investigate the cognitive precursors of movement.

2.2 Introduction

Williams et al. (1998) is the key paper for this thesis and the basis for all methods used here. Therefore, before adopting their procedures for our experimental protocol, it will first be necessary to replicate their main findings. Williams et al. (1998) asked subjects to detect the presence of weak electrical shocks delivered to the right index finger when the finger was moving or at rest. The moving finger showed time-dependent reductions in the proportion of stimuli detected (Chapter 1; Figure 1.2). In order to investigate the spatial characteristics of sensory suppression, subjects were asked to detect the presence of weak electrical shocks delivered to various sites on the body. During movement there was almost complete suppression of detection at the sites closest to the moving digit. However, Williams et al. (1998) found a decreasing effect of sensory suppression with distance. Thus, when the shock was delivered to the contralateral hand, a small (approximately 10%) non-time-dependent decrease in detection was observed. This decrease was attributed to the attentional demands placed on the subjects who were asked to divide their attention between the motor task (finger abduction) and the perceptual task (detection of weak shock stimuli). In short, Williams et al. (1998) showed that

movement-related decreases in detection of near-threshold stimuli can precede movement onset and the onset of EMG activity demonstrating premovement sensory suppression. They also demonstrated that sensory suppression is focused i.e. both the time-course and amplitude of the modulation were dependent on the distance between the stimulation site and the body part in motion.

Some aspects of the current procedure were more strongly controlled than in the original (Williams et al., 1998) procedure. Modifications included the following: i) the movement (finger abduction) was fixed at 20° of angular rotation whereas in the original study subjects were free to abduct their finger any angle between 15 and 45°; ii) a 'response window' of 700 ms was introduced in order to encourage consistent movements and facilitate targeting of the shock timing in the interval just before EMG onset and iii) in the present study shocks were delivered to the hairy dorsum of the index finger as opposed to the glabrous skin of the finger-pad. These changes are discussed in more detail in the Methods section. Using this procedure, we aimed to replicate the main findings relating to the timing, magnitude and spatial extent of sensory suppression. A shock was delivered to the right index finger during interleaved blocks of right and left index finger movements.

In line with the original study, it was predicted that shock detection rates would be significantly lower prior to and during movement relative to when the finger is at rest, thereby demonstrating movement-related sensory suppression. For the right index finger, the time-varying decrease in detection rates with worst shock detection just after EMG onset was predicted. In Williams et al. (1998), the left hand also showed approximately 10% sensory suppression during movement with no temporal gradient in detection rates prior to EMG onset. Data analysis for sensory suppression effects will focus on movement trials when the shock is delivered prior to EMG onset as suppression in this interval is interpreted as evidence that central signals related to the preparation and execution of the movement are responsible for sensory suppression. Such a result would validate the paradigm as a means of studying premovement, motor-sensory interactions.

2.3 Methods

2.3.1 Subjects

A total of 20 naïve paid subjects participated in this study. Subjects who showed a difference of 15% or greater between pre- and post-experiment sensory detection threshold levels were excluded from the analysis (see Section 2.3.2.3). Four subjects were thus excluded. Data from the remaining 16 subjects were analysed. The mean age was 34.4 (SD=3.8) years. One subject was left-handed for writing and 15 were right-handed. The experimental protocol was approved by the University College London ethics committee.

2.3.2 Procedure

Data from each subject were gathered in a single session lasting approximately 1 hour. At the beginning of the session, subjects received verbal and written instructions about the motor and perceptual tasks they were to perform and were familiarised with the equipment. The motor and perceptual tasks are now described.

2.3.2.1 Motor Task

The motor task consisted of a simple reaction time (RT) task whereby subjects made a rapid abduction of the right or left index finger as quickly as possible after the presentation of a visual Go signal. Left and right finger movements were tested in separate blocks. The Go signal was a green LED, mounted in a black box positioned centrally on a small table, approximately 1 m in front of the subject in the midline (see Figure 2.1).

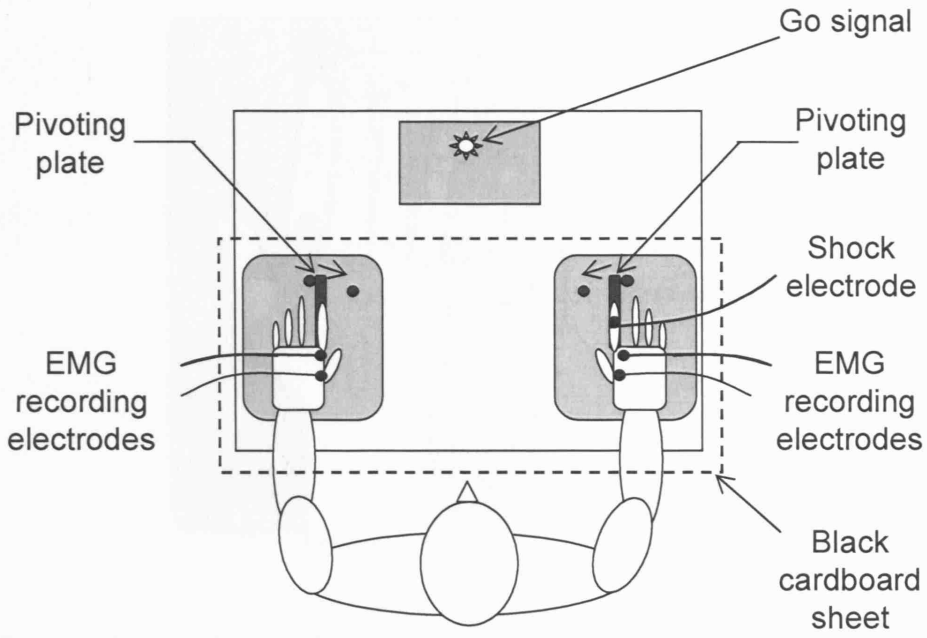

Figure 2.1. Experimental set-up. Subjects placed their two hands on mouse-pads with their index fingers resting on a pivoting plate. The Go signal was presented via an LED positioned in the midline, approximately 1 m in front of the subject. A black cardboard sheet (dashed rectangle) was placed over the subject's hands and lower arms to prevent direct vision. Note that the shock electrode is mounted on the right index finger only.

Subjects were seated in a comfortable chair in front of a table. Both arms rested on cushioned armrests. Each hand was positioned on a computer mouse-pad. Each index finger rested on a pivoting plate (1.8 x 10 cm) the base of which was aligned with the axis of rotation of the index finger's metacarpophalangeal joint (Figure 2.1).

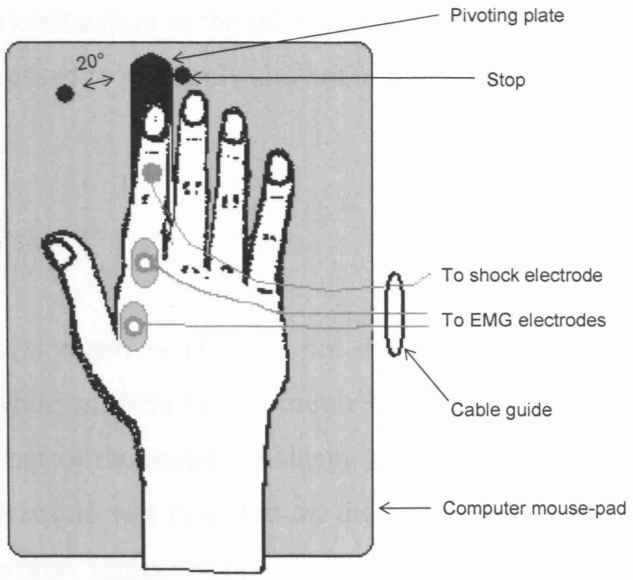

Figure 2.2. Set-up for the right hand after Williams et al. (1998). The set-up for the left hand was the same (mirror image) but with no shock electrode. Subjects placed their hand on a computer mouse-pad with each index finger resting on a plastic, slightly concave pivoting plate. Angular displacement of each index finger was measured using a potentiometer (not shown) integrated into the pivoting plate that supported each index finger. The finger is shown in the start position (potentiometer output = 0°). During a movement trial, upon presentation of the Go signal, the subject quickly abducted their finger (20°), still resting on the pivoting plate, 'over and back' as shown by the small double-ended arrow.

The distance between centres-of-rotation for the right and left finger pivoting plates was 36 cm. The subject was asked to sit as comfortably as possible and to relax their index fingers in the start position (0° angular displacement). Here, the amplitude of finger movements was more strongly controlled than in the Williams et al. (1998) study. Their subjects' movements could be of any amplitude greater than 15°, which may have resulted in differences in movement styles within test runs and between subjects. Here, all finger movements were limited to 20° amplitude. The subjects were requested to abduct one of their index fingers so that the top of the pivoting plate touched the stop post and then return to the start position (0°) as quickly and consistently as possible. Pilot studies showed that subjects found finger abductions within this range natural and easy to perform. During practice, subjects were trained to make consistent and fast movements, thereby further minimising differences. Subjects were also requested to only move the index finger and to maintain the rest of the hand as steady as possible during trials in

order to limit muscle contraction to the relevant agonist muscle (first dorsal interosseous, FDI). Subjects were asked to sit calmly and not to move their body during trials.

2.3.2.2 Perceptual Task

Subjects were asked to report whether or not they detected the occurrence of a weak electrical stimulus while performing the motor task and at rest. Here, the shock was delivered to the dorsum of the middle phalange of the right index finger whereas in the original study the electrode was mounted on the finger pad of the index finger. Other studies have demonstrated sensory suppression with electrical stimuli e.g. to the median nerve (Pertovaara et al., 1994; Feine et al., 1990; Chapman et al., 1987; Milne et al., 1988). However, it is not known if sensory suppression is obtained when electrical stimuli are delivered to the dorsum of the index finger in humans. Sensory gating has been observed in monkeys when electrical stimulation was applied to the dorsum of the finger but no sensory suppression effects were observed when the finger pad was stimulated (Jiang et al., 1990). The shock electrode on the right index finger was clearly pointed out to subjects at the start of the experiment so as to avoid any confusion regarding the source of the shock as EMG electrodes were also mounted on the back of each hand (see Figures 2.1 and 2.2). No information regarding the proportion of trials with and without a stimulus was given to the subjects. No feedback was given with regard to the accuracy of the subjects' perceptual judgements. The sensory stimulus consisted of a square-wave pulse generated by a Stanmore neuromuscular stimulator (adapted for research purposes by University College London Institute of Neurology, Sobell Research Department). The stimulus was delivered via a single bipolar, lightweight, custom-built, Teflon surface electrode (4 mm in diameter; see Figure 2.3).

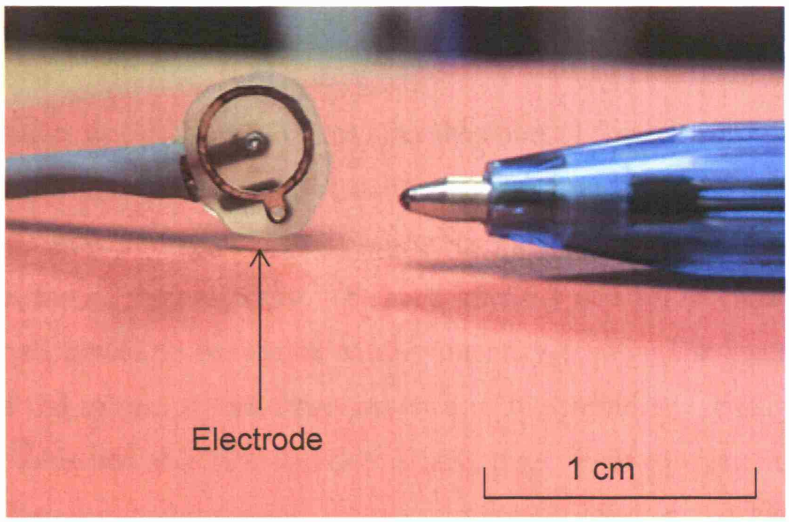

Figure 2.3. Photograph of custom-built bipolar electrode, scaled for size against the nib of a ballpoint pen. The electrode was mounted on the dorsum of the right index finger.

Before applying the electrode, skin resistance was minimised and electrode adhesion maximised by vigorously cleansing the electrode sites with surgical spirit. The electrode was attached to the finger using surgical tape; tight enough to hold the electrode firmly in place but loose enough so as not to cause any discomfort (Schmidt, 1961). Care was taken that wires from the electrodes could not brush against the hands during trials. All wires were supported above and away from the hands using a cable support (see Figure 2.2). The stimulus consisted of a square-wave pulse of 10 mA fixed amplitude, delivered by a portable 6 Volt neuromuscular Stanmore stimulator custom-built by UCL Institute of Neurology (Sobell Research Department). Perceived intensity of the stimulus was adjusted by varying the pulse-width. At the beginning and end of each experiment, a simple staircase procedure of approximately 40 trials (Levitt, 1971) and with the subject's finger at rest was used to establish the shock intensity level (range 17 - 27 μs i.e. microseconds) for the experimental blocks.

2.3.2.3 Staircase procedure

The shock stimulus was delivered 200 ms after the onset of a green LED signal. Subjects were informed that the shock would be delivered almost immediately after the onset of the LED signal. Intensity was varied by adjusting stimulator pulse-width in single steps (1 μs). At the start of the procedure, the shock intensity was set at 15 μs. None of the subjects reported detecting the shock at this intensity level. The intensity was then increased until the subject reported the presence of the stimulus; typically about 20 μs. The shock stimulus was then repeated at this level three times to ensure the subject was familiar with the nature of the shock. Subjects reported whether they felt the shock by responding 'yes' or 'no'. If the subject responded 'no' for one or more of these trials the shock intensity was increased. When the subject responded 'yes' for 3 successive trials, the shock intensity was reduced in single steps until the subject was unable to detect the stimulus for 3 successive trials. The intensity was then increased again until the subject reported the presence of the shock for three successive trials. This up-down procedure was repeated a minimum of 3 times and was used to establish the lowest shock intensity at which approximately 90% of stimuli delivered to the resting finger were detected. This level was then used as the intensity level for the rest of the experiment (see also Section *2.3.2.2)*. The staircase procedure was repeated at the end of the experiment. The pre-experimental staircase procedure was followed by 2 practice blocks and 4 experimental blocks of 44 trials.

2.3.2.4 Data Collection

The experiment was run using LabView software. Three trial types were presented. Each trial was initiated by the experimenter and lasted for 1700 ms (Figure 2.4). On 'movement' trials (N=30 per block), subjects were verbally instructed at the start of the trial to move when the Go signal was presented. These trials were used to gather data on the effects of movement on the perception of the shock stimulus. On 'non-movement' trials (N=10 per block), subjects were instructed verbally not to move during the trial, and

the shock stimulus was applied to the non-moving finger. These trials monitored the perceptual performance with the finger at rest. Finally, catch trials (4 trials per block), with no shock stimulus were divided equally between movement and the non-movement trials. The different types of trials were intermixed randomly and the order of presentation was randomised anew for each block. Occasionally during the practice block, the shock intensity was fine-tuned but then maintained at this level for the remainder of the experiment.

Before each trial, the experimenter issued a verbal instruction ('move' or 'don't move'). An acoustic warning signal then marked the start of the trial (Figure 2.4). After a fixed foreperiod of 1000 ms, the Go signal was presented for 700 ms.

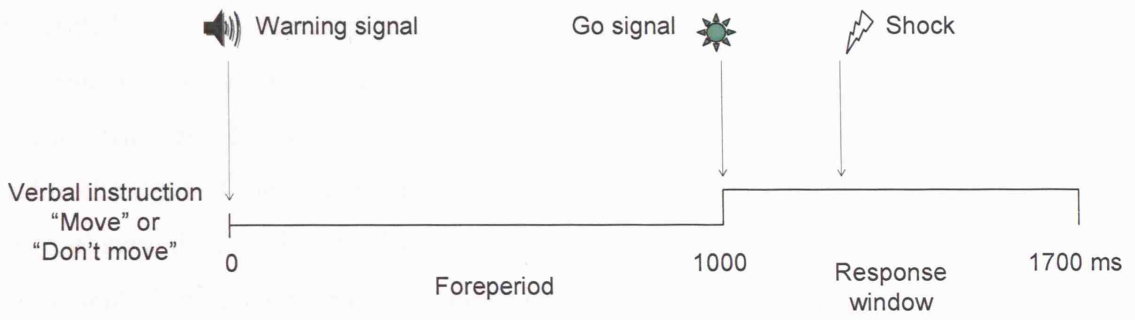

Figure 2.4. Experimental design for Experiment 1. The experimenter gave a prior instruction before the start of each trial, either 'move' or 'don't move'. An acoustic warning signal then marked the start of the trial. The Go signal was presented 1000 ms later. Subjects either moved or did not move their index finger in accordance with the prior instruction during the response window (i.e. the interval from 1000 to 1700 ms). The shock was delivered 50 ms before each subject's mean RT on movement trials.

This time period (700 ms) acted as a response window encouraging fast and consistent reaction times, and facilitating the delivery of the shock in the critical time period prior to movement. To concentrate sampling around the onset of movement, each subject's mean RT i.e. the mean difference between the onset of the Go signal and the onset of EMG activity was estimated from a practice block. The timing of the sensory stimulus during the experimental blocks was 50 ms before each subject's mean RT, as measured from the

practice block. Williams et al. (1998) reported that the critical period for centrally-mediated sensory suppression is 0-120 ms before the onset of EMG activity. Natural trial-to-trial variations in RT resulted in the collection of an adequate sample of perceptual ability during a period 200 ms prior to movement to 50 ms post movement. At the end of each trial, subjects reported verbally whether or not they had perceived a shock stimulus (by responding 'yes' or 'no'). Their response was entered into the computer by the experimenter and stored with each trial. Subjects were not informed about the proportion of trials in which a shock stimulus was presented. After each trial, the experimenter could view the trial information on a computer screen and could monitor any errors in performance. The experimenter then initiated the next trial after an intertrial interval of at least 1 second.

Angular displacement of each index finger was measured using a potentiometer integrated into the pivoting plate that supported each index finger. In this way the movement trace could be inspected on the computer screen by the experimenter at the end of each trial (see Figure 2.5 for illustrative trial). The experimenter was seated behind and to the right of the test apparatus and could not be seen directly by subjects. Subjects were observed carefully by the experimenter to ensure that there was no superfluous movement during each trial. Electromyographic and potentiometer activity was also monitored on screen by the experimenter after each trial. Any trial during which the subject produced movements unrelated to the motor task, as well as any trial after which the subjects reported distraction or discomfort or lack of readiness, was noted and later rejected from the analysis. The EMG activity of the first dorsal interosseous (FDI) muscle was recorded via 6 mm diameter surface electrodes placed 25 mm apart (centre to centre) on the skin overlying the muscle with the reference electrode positioned on the index finger knuckle (see Figure 2.2). The earth electrode was placed on the back of the wrist next to the ulna sternoid. The EMG signal was amplified and sampled at 1000 Hz. All data were stored for later offline analysis.

2.3.2.5 Data analysis

Offline, each trial was inspected visually. For each movement trial the onset of EMG was determined by positioning the crosshairs of the cursor over the point where EMG activity first began a sustained increase above baseline (Figure 2.5). The reaction time for each movement trial was recorded in this way. Approximately 1% of trials were discarded at this stage, usually because EMG activity was observed simultaneous with the Go signal indicating anticipation of and not reaction to the signal. Because performance during movement was to be compared with performance at rest, as this comparison gives the magnitude of sensory suppression, it was very important that perceptual performance during immobile trials be constant throughout the experiment. To verify this, the staircase procedure was repeated at the end of the experiment for each subject and performance was compared using t tests.

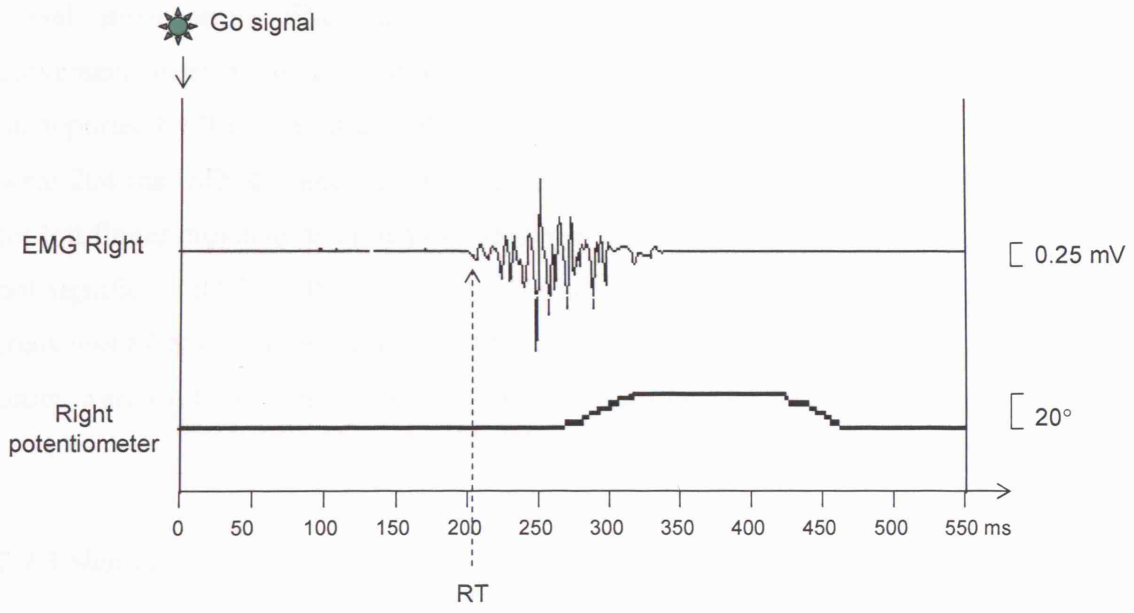

Figure 2.5. Illustrative trial showing EMG recordings and movement potentiometer output (limited to 20°) for a single subject performing a right index finger abduction. Reaction time (RT) was defined as the onset of EMG activity.

2.4 Results

2.4.1 Stability across time of perceptual performance at rest

For each subject, pre-experiment threshold levels to the finger at rest were compared with post-experiment stimulus levels. The pre- and post-experiment staircases for shock intensity thresholds did not differ (mean pulse-widths= 20.3 μs) suggesting that perceptual performance during immobile trials was constant throughout the experiment.

2.4.2 Reaction times

Figure 2.5 shows an example of an illustrative trial showing the movement trace and EMG records from a single illustrative trial from one subject. Reaction time was defined as the onset of EMG. Detailed inspection of a subset of 3 subjects' trials showed that first dorsal interosseous EMG activity, the principal agonist, preceded potentiometer movement onset by an average of 60 ms; this value is somewhat greater than the 35-55 ms reported by Williams et al. (1998). Reaction times for left and right finger movements were 204 ms (SD=41) and 215 ms (SD=50) respectively. Though there was a tendency for left finger movements to be faster than the right finger movements, this difference was not significant $t(15)=1.487$; $p=0.158$. The percentage of pre-EMG onset (premovement) trials was 84.8%. The remaining 15.2% of trials, in which the shock occurred after EMG onset, were excluded when analysing premovement sensory suppression effects.

2.4.3 Shock detection

First, all movement trials are analysed together in order to see if we replicated the original study (Williams et al. 1998). Then, the 84.8% of movement trials where the shock was delivered before EMG onset were analysed separately in order to isolate centrally-mediated sensory suppression effects.

2.4.3.1 All movement trials

Shock detection rates on the moving right finger were 38% (SD=23) and 84% (SD=15) for the stationary right finger. Shock detection rates on the right finger were 59% (SD=24) when the contralateral left finger moved. When the instruction was not to move, shock detection rates for the contralateral finger were 90% (SD=11). The data were subjected to a 2 x 2 ANOVA for the factors hand ('left' vs. 'right') and trial type ('movement' vs. 'non-movement'). There was a main effect of hand $F(1,15)=8.421$; $p=0.011$ and a main effect of trial type $F(1,15)=65.596$; $p<0.0001$. The interaction was also significant $F(1,15)=7.027$; $p=0.018$. Greenhouse-Geisser adjustments to the degrees of freedom were applied where appropriate, and corrected p-values are reported. Post-hoc t tests were used to explore this interaction. Detection rates for shocks on the right finger were significantly lower when the right hand moved than when the left hand moved $t(15)=3.099$; $p=0.007$, providing evidence for the effector-specific nature of sensory suppression. Sensory suppression is greater on the moving limb. There was no difference in shock detection rates between hands for non-movement trials $t(15)=1.559$; $p=0.140$.

2.4.3.2 Movement trials where the shock was delivered prior to EMG onset

In order to focus our analysis on centrally-mediated sensory suppression, we analysed movement trials where the shock was delivered prior to EMG onset (and before any peripheral feedback could have occurred) separately i.e. removing the 15.2% of trials where the shock occurred after EMG onset. Pre-EMG shock detection rates during left finger movements were 60% (SD=24) and 90% (SD=11) when the finger was at rest (see Figure 2.6). Detection rates during right finger movements were 43% (SD=22) and 84% (SD=15) during non-movement trials. A 2 x 2 ANOVA for the factors hand ('left' vs. 'right') and trial type ('movement' vs. 'non-movement'), revealed a main effect of hand $F(1,15)=8.421$; $p=0.011$ and a main effect of trial type $F(1,15)=55.906$; $p<0.0001$. The interaction was also significant $F(1,15)=6.496$; $p=0.022$.

Post-hoc t tests revealed that shock detection was significantly better when the finger was stationary than when moving for the left $t(15)=4.804$; $p<0.0001$ and the right hand $t(15)=9.679$; $p<0.0001$. Furthermore, significantly more shocks were detected prior to movement on the left hand than on the right hand $t(15)=3.196$; $p=0.006$. However, as reported above (Section *2.4.3.1*), there was no difference between the hands during non-movement; $p=0.140$.

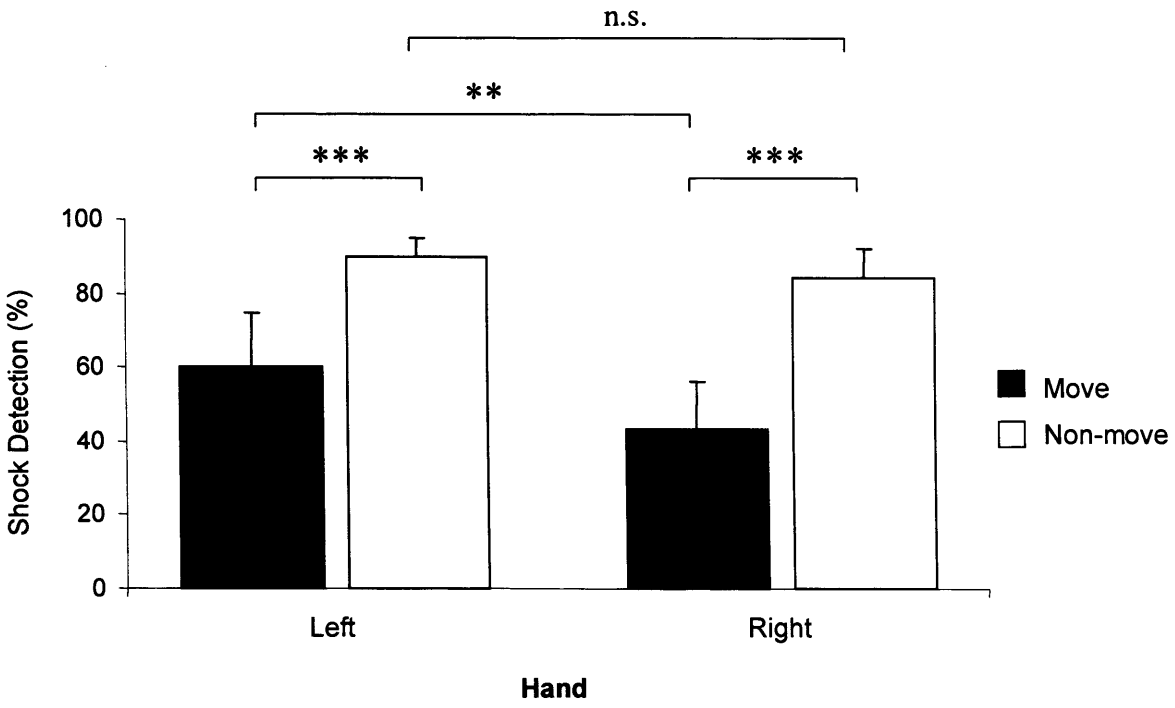

Figure 2.6. The mean percentage of shocks (error bars +/- SD) detected for non-movement and movement trials when the shock was delivered prior to EMG onset, for the left and right hands.

Table 2.1 summarises and compares the global shock detection performance of all subjects in the three trial types (movement; non-movement and catch trials) for the current study and the original version (Williams et al., 1998).

Table 2.1. Shock detection rates during movement and non-movement trials for the left and right index fingers for the present study are compared with data from the original study (Williams et al., 1998) given in italics. For the "present study", only movement trials where the shock was delivered before EMG onset are given.

	Trial Type		
	Movement (%)	**Non-movement (%)**	**Catch trials (%)**
Right index finger			
Present study	43	84	3.1
Williams et al. (1998)	*40*	*94*	*0.0*
Left index finger			
Present study	60	90	1.6
Williams et al. (1998)	*84*	*95*	*0.1*

Of the 256 catch trials (no shock stimulus), only 12 false positive responses were noted for the left and right hands, indicating that subjects used a conservative response strategy throughout the experiment. In the original study, Williams et al. (1998) also reported a low number of false positives, 12 false positive responses out of 1,479 catch trials, after pooling the data from a larger number of experiments. In the current study, there was no significant difference in the number of false positives between the left and right hand conditions; $t(15)=1.464$; $p=0.164$, indicating no change in response bias between hands (Table 2.2).

Table 2.2. The number of false positive catch trials pooled across subjects during movement and non-movement trials for left and right hand movements. The shock was delivered to the right hand only.

Trial Type	**Left hand**	**Right hand**
Movement	0	4
Non-movement	4	4

The number of errors of commission (when subjects moved their index finger when the instruction was not to move) was 1.1%. Again there was no significant difference in the number of errors of commission between the left and right hand; p=0.774. Comparisons between performance on the movement and non-movement trials showed that significantly fewer stimuli were perceived during movement by all subjects for both hands. As can be seen from Table 2.1, the results are broadly consistent with the original findings by Williams et al. (1998). Movement attenuates perception of shocks to the moving body-part. However, an unexpected finding was the 30% (90-60=30; Table 2.1; "left index finger", "present study") reduction in detection rates for the contralateral (left) index finger which is considerably higher than the 11% (95-84=11; see Table 2.1; "left index finger", "Williams et al., 1998") found in the original study; a finding that will be discussed in more detail in the Discussion (Section 2.5).

2.4.4 Time-dependent changes in the detection of stimuli applied to the moving digit

To study the time-course of any variation in performance during movement, trials occurring within 200 ms prior to EMG onset to 50 ms post EMG onset were grouped into 50 ms bins relative to EMG onset. The proportion of stimuli perceived was calculated for each bin. These proportions were then compared with the proportion of stimuli perceived during the corresponding immobile trials.

By calibrating stimulus intensity relative to a given detection level, approximately 90% detection with the finger at rest, baseline detection performance at rest was identical and comparable from subject to subject. This permitted the pooling of data from multiple subjects. Pooling all the data yields a general curve allowing time-dependent changes in the detection of stimuli applied to the moving digit to be revealed (Figure 2.7). However, it should be noted that not all subjects necessarily contribute to each data point, particularly near the edges of the graph. While pooled data gives the overall picture, the data in Figure 2.7 were not analysed as repeated measures. Statistical analyses for

sensory suppression effects were only carried out as repeated measures using subjects' individual mean scores.

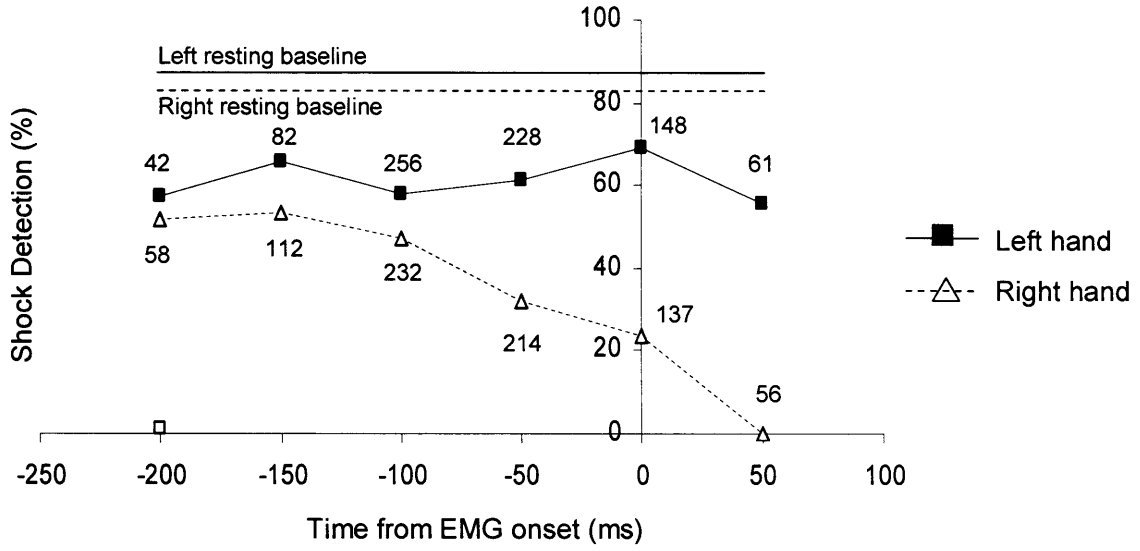

Figure 2.7. Effects of left and right index finger movement on the ability of 16 subjects to detect fixed intensity stimuli. Detection performance over time is plotted relative to the onset of EMG (0 ms). The data are pooled for all subjects. The values displayed next to each data point on the curves refer to the number of trials contained in that data point.

Figure 2.7 shows that the effects of right index finger movement on the ability of sixteen subjects to detect the weak shock stimulus to the moving finger were not uniform over time. For the right hand, performance declined beginning 100 ms before EMG onset with the peak decrease in perceptual performance coinciding approximately with movement onset i.e. 50 ms after EMG onset. Furthermore, a linear regression equation with a slope significantly different from zero ($p=0.005$), indicated that for the right hand there was a time-dependent change in performance. A different pattern of perceptual modulation was observed when the left finger was abducted (Figure 2.7). The slope for the left hand was not significantly different from zero ($p=0.894$) indicating no time-dependent change in performance. This finding replicates the original study; while there is a relatively small and significant decrease in perceptual performance for a more distant body part, it does

not seem to be time-dependent. Indeed, the small but time-invariant sensory suppression on the left hand may reflect the diversion of attention from the shocked right hand to the active left hand.

While Figure 2.7 shows a smooth time-dependent decrease in shock detection rates prior to EMG onset, there is no evidence of a continuous change in sensory suppression *for each subject*. It might equally be an all-or-none suppression with varying timing, within and between subjects, which gives rise to the smooth function. Therefore trials occurring from 100 ms to EMG onset were sorted into 50 ms bins (i.e. 100 ms; 50 ms and 0 ms) for each subject. Subjects who contributed 5 or more trials to each time-bin were included. Thus 5 of the 16 subjects were excluded. Using linear regression, the slope value was then calculated for each of the remaining 11 subjects. A decrease in shock detection rates with time in line with the original study (Williams et al., 1998) was predicted. A one-tailed, one-sample t test compared against zero confirmed a significant time-dependent decrease in shock detection rates [$t(10)=2.451$; $p=0.018$] suggesting a continuous, progressive onset of sensory suppression.

2.5 Discussion

The purpose of this study was to replicate the procedures and main findings of the finger abduction and perceptual motor task first described by Williams et al. (1998). The present results broadly replicated the original findings by showing that movement-related decreases in detection of near-threshold stimuli can precede the onset of EMG activity and movement onset. We extend the previous findings to the dorsum of the moving digit in humans. Detection of shock stimuli delivered to the moving right finger diminished significantly and in a time-dependent manner with the first decrease occurring approximately 100 ms before the onset of EMG activity (Figure 2.7). Modulation observed prior to EMG onset is thought to reflect centrally-mediated sensory suppression (Williams et al., 1998; Williams and Chapman, 2000; 2002). After EMG onset, there was

almost complete suppression of detection at the moving digit, presumably due to masking.

In contrast, the contralateral left hand showed no time-dependent decrease in perceptual performance, consistent with Williams et al. (1998). The only discrepancy with the original results arose from an unexpected deeper suppression for the contralateral hand during movement (30 %; see Results) compared to that reported by Williams et al. (11%; 1998). It is not clear why attenuation for the contralateral hand was deeper in the current study. One possibility is that the shock was delivered to the hairy dorsum of the finger in contrast to the glabrous skin of the finger pad in the original study. It might be argued that the less dense innervation and differing receptor types of the dorsum may contribute to different processing of the stimulus yielding a variation in sensory suppression. However, we do not think this is the case. Other studies e.g. Pertovaara et al. (1994) also used electrical stimulation to the median nerve of the arm and found only 12% attenuation on the contralateral limb. A more plausible explanation is that the difference in the depth of modulation for the contralateral hand stems from differences in the methodologies used. Williams et al. (1998) tested the left and right hands in separate experiments using different subjects. In contrast, in the current study we tested both hands for each subject using interleaved blocks within the same experiment.

2.5.1 Sources and mechanisms of the movement-related decrease in tactile detection

The time-dependent characteristics of the movement-related decrease in detectability (Figure 2.7) help to define the necessary attributes of the gating mechanism(s). Both top-down, centrally-mediated inhibition and / or peripheral masking sources may have contributed to generating the observed time-dependent gradient. Strong evidence in favour of a central source for sensory suppression is the observation that at the moving digit, detection began to decline 100 ms before the onset of EMG activity i.e. well before any peripheral feedback could have been generated. This early time-dependent decrease is thought to be related to the preparation and execution of the movement (Williams et al.,

1998, Williams and Chapman, 2000; 2002; Chapman and Beauchamp, 2006; Coulter, 1974; Ghez and Lenzi, 1971; Jiang et al., 1990). Other spatial and temporal characteristics argue in favour of a role for peripheral masking sources in mediating the movement-related gating. The timing of the worst sensory detection at the right index finger coincided with the onset of movement; approximately 50 ms after onset of EMG activity (Figure 2.7). It is reasonable to assume that peripheral feedback commences at this moment e.g. muscle spindle discharge, and that this feedback contributed to the sharp decrease in detection performance by masking cutaneous afferent signals at this time.

2.5.2 Methodological considerations

The electrical stimulation used in this study presents a reliable means of providing stimuli of identical duration and detectability. The stimulating electrode used was extremely light and therefore unlikely to affect movement characteristics. The shock would mainly activate cutaneous afferents. Subjects reported that the electrical stimuli did not feel unnatural, just like a 'very light tap', and 'very weak'. By calibrating stimulus intensity relative to a given detection level (approximately 90%), baseline detection performance at rest was identical and comparable from subject to subject. This permitted the pooling of data from multiple subjects.

Several alternative explanations of the findings were considered and dismissed. First, movement of the index finger may have dislodged the stimulating electrode and thereby reduced the proportion of afferent fibres stimulated by the shock. This explanation is unlikely for several reasons. First, the electrode design helped prevent dislodgement; the anode of the electrode consisted of a small point which gripped the skin on the back of the finger, leaving a small mark. At the end of the experiment the skin was examined during electrode removal to verify that only one mark was present on the skin. Second, thresholds were measured both before and after the experiment and were compared statistically, thus ensuring that there were no systematic changes in shock detection rates or drift over the duration of an experiment.

An important consideration is whether the presence of the shock affected preparation for the ensuing movement. There may have been a differential trade-off between motor preparation and shock detection as subjects allocated limited attentional resources to the detection of the shock at the expense of preparation for movement. Therefore, this may have resulted in the observed time-varying detection rates for the right shock stimulus hand and the absence of time-varying detection rates for the left hand where no shock stimulus was applied (see Figure 2.7). It is known that movement-related gating is a function of the kinematics of the movement, with faster movements producing larger gating effects (Angel and Malenka, 1982).

It could be argued that the current design should include checks to verify that there was no difference between the left hand and the right shock-stimulus hand for kinematic parameters, such as peak amplitude, peak velocity and peak acceleration. Here, we did not examine kinematic factors as this has been done elsewhere (see Williams et al., 1998) and no significant differences were found (see also Section 11.3.1). Furthermore, for the current experiment, EMG profiles and potentiometer outputs for the left and right hands for each subject were visually inspected and no obvious differences were evident.

An advantage of the movement performed in this study, abduction of the index finger, is that it has only one major agonist (the FDI muscle). Other intrinsic and extrinsic hand muscles are likely to show small amounts of co-contraction; however these effects are likely to be minimal as the movement amplitude was controlled at 20°. First dorsal interosseous EMG activity thus represented the earliest peripheral response to the motor command, giving a clear time-point for data analysis and interpretation of the results. Practice and fatigue effects did not appear to play a significant role on perceptual performance during these experiments, as confirmed by the staircase procedure. Subjects appeared able to detect signals at approximately the target rate of 90% at rest while minimising false positives. The rate of false positives was always very low, a good indication that bias was not a significant factor in the experimental results. Thus, subjects appeared to perform near optimally.

In the current experimental design shock stimuli were delivered on 90% of movement and non-movement trials. In such a design, subjects may exhibit a response bias i.e. a willingness to report the presence of a shock stimulus, which might systematically change in relation to the motor task (movement vs. non-movement). In order to control for such a bias, Chapman and Beauchamp (2006) included equal numbers of trials with and without a shock stimulus. This allowed them to evaluate the perceptual data from individual subjects using two methods. The first was the 'proportion of stimuli detected' method as used for the experiments in this thesis. The second method was a more bias-free method based on signal detection theory (SDT; Green and Swets, 1988) using the index of detectability (d'). For the SDT approach, the data were used to construct a receiver operating characteristic curve from which d' was derived.

Chapman and Beauchamp (2006) found that the two methods yielded virtually identical results thereby confirming that changes in bias did not contribute to the results. Both measures of detection, d' and the "proportion detected" method revealed the time-dependent decrease in shock detection rates that began prior to the onset of movement. Values obtained for non-movement trials were also similar. They noted that the "proportion detected" method was slightly more sensitive at detecting small changes in the timing of sensory suppression and offered this advantage over the d' method. A disadvantage of SDT is that only 50% of trials contain a shock stimulus. Many more trials are required in order to measure a sensory suppression effect of a given size. The SDT method therefore would result in experiments that are considerably longer thereby increasing the possibility of fatigue effects. Taking all into account, here it was decided to use the "proportion detected" method. This method yields similar results to less conservative more bias-free accounts such as d'. It also comes with the benefit of greater sensitivity for detecting small changes in the timing of sensory suppression while avoiding longer experiments with the associated possibility of fatigue effects.

Controlling movement amplitude at 20° is likely to ensure that movements, and hence related sensory suppression effects, are relatively invariant. One concern however, was that exafference created by the contact of the pivoting plate against the stop (see Figure

2.2) during movement might have produced unwanted masking effects. This does not seem to have been the case, as our findings are broadly in line with previous results (Williams et al. (1998; Williams and Chapman, 2000; 2002; Chapman and Beauchamp, 2006). Furthermore, as our analysis for sensory suppression effects was confined to trials where the shock is delivered prior to the onset of EMG and movement, it seems that any exafferent effects caused by the stop were negligible.

In conclusion, we have replicated the findings of Williams et al. (1998) showing that sensory suppression is stronger and earlier on the moving limb, in line with the proposal that central mechanisms are at play. We can now use the current methodology, hereafter referred to as the "sensory-detection task", or the "S-D task", as a sensitive and replicable probe of motor and sensory interactions to further explore the questions laid out in Chapter 1. We start by exploring the role of preparation and prior information for sensory suppression, the topic of the next chapter.

Chapter 3: Sensory suppression and cognitive-motor preparation

3.1 Abstract

Subjects prepared a motor response, and then either executed it in response to a subsequent Go signal, or cancelled the movement if a NoGo signal occurred. Subjects had to detect weak shocks which were delivered after the Go/NoGo signals on some trials. Results were compared to the *prior instruction* task (Experiment 1 and Williams et al., 1998) in which subjects knew at the start of the trial if they should move or not. We found that detection rates on movement trials were lower than on non-movement trials, consistent with sensory suppression. There was no difference between tasks in detection for movement trials. Clear premovement sensory suppression was present for both tasks. Crucially, detection rates for non-movement trials were significantly lower in the Go/NoGo than in the *prior instruction* task, suggesting an element of sensory suppression associated with actions which are prepared, but then inhibited before execution. The result is discussed in the context of a premotor origin of sensory suppression.

3.2 Introduction

All motor control models assume that making a voluntary motor response to a sensory signal involves a number of central information processing stages that culminate in the activation of the muscles (Crammond and Kalaska, 2000; Donders, 1969; Requin et al., 1988; Riehle et al., 1994; Rosenbaum, 1983; Sternberg, 1969; Wolpert, 1997). It is also generally assumed that at least some of these sensorimotor processes are serially ordered and are grouped into two broad sequential stages of preparation and execution. Neurophysiological studies have shown that while sequential models may be simplistic, they have heuristic value for describing the general information processing structure of cortical motor control mechanisms and generate useful, testable predictions (Andersen et al., 1997; Crammond and Kalaska, 2000; Georgopoulos et al., 1986; Riehle et al., 1994). A key distinction in such models is the division between motor preparation and motor execution. Preparation involves planning how to move, assembling the motor command and adjusting the body's state for the impending movement. Execution involves physical contraction of muscles. The moment that a volley of action potentials begins its descent from motor cortex to the muscle is a useful dividing point between preparation and execution. Preparation can be initiated by the sensory signal but is not causally related to muscle activation and so is theoretically dissociable from overt motor output (Lecas et al.,

1986; Rosenbaum, 1983). In the *prior instruction* task, the verbal instruction given before the start of a trial provides definite information about a movement whose execution must be delayed until the subsequent Go signal. This procedure should allow subjects to prepare the parameters of the ensuing movement in advance and therefore reduce the amount of information processing needed after the Go signal. If the prior instruction is not to move, then subjects simply have to do nothing i.e. no preparation is required.

As outlined in Chapter 1, modulation that precedes the onset of movement and electromyographic (EMG) activity has been interpreted as evidence that central signals, related to the preparation and execution of the movement, play a role in sensory suppression (Chapman et al., 1988; Coulter, 1974; Dyhre-Poulsen, 1978; Ghez and Lenzi, 1971; Jiang et al., 1990). However, humans can prepare an action but then not execute it, either because they change their mind, or because an external signal tells them to cancel execution. In the laboratory, the Go/NoGo task is used to investigate these situations (e.g. Hoshiyama et al., 1996). In the Go/NoGo task, subjects respond to a prepotent Go signal (e.g. 75% probability) but withhold the prepared response when an occasional NoGo signal (e.g. 25% probability) occurs. However, the absence of any overt behaviour on NoGo trials has limited scientific study of what happens when movement execution is cancelled.

Here, we have compared sensory attenuation on movement and non-movement trials to investigate processes of movement preparation and cancellation. We combined the sensory suppression paradigm (Experiment 1; Williams et al., 1998) with the Go/NoGo paradigm. In the *prior instruction* task, each trial was preceded by a verbal instruction (to move or not to move). Shock detection rates for movement and non-movement trials were compared with a Go/NoGo version of the same task. In the *prior instruction* task, subjects knew in advance whether they would move on any given trial, and thus there was no need to prepare actions on non-movement trials (see Table 3.1). In contrast, in the Go/NoGo task, subjects had to prepare a motor command and wait for a signal and process the information conveyed by that signal, before deciding whether to execute the

movement or cancel it. If sensory suppression is indeed linked to preparatory processes, then it is reasonable to expect some sensory effects on NoGo trials because a response is prepared but then cancelled prior to execution.

Table 3.1. Time-line of events for the *prior instruction* and Go/NoGo tasks. In the *prior instruction* task the subject receives a verbal instruction before the start of each trial, and therefore knows with certainty whether to move or not. However, on the Go/NoGo task where there is no verbal instruction at the start of the trial, the probability of movement on every trial is 75% (Go: NoGo ratio = 3:1). Therefore, subjects must prepare to move on every trial.

Task	Trial	Verbal instruction	Movement probability	Motor response	Hypothesised level of preparation
Prior instruction	Movement	"Move"	100%	Execute movement	Prepare
	Non-movement	"Don't move"	0%	Do nothing	Do nothing
Go/NoGo	Go	None	75%	Execute movement	Prepare
	NoGo	None	75%	Inhibit movement	Prepare

3.3 Materials and Methods

3.3.1 Subjects

Thirteen paid subjects took part with local ethical committee permission. The data from 3 subjects were excluded because their detection of cutaneous shocks at rest was unstable across the experiment (post-test detection varied more than +/-15% from pre-experiment levels; see later). Data from the remaining 10 subjects (4 female, 7 right-handed, mean age 33 years) were included in the final analysis.

3.3.2 Procedure

The procedure was based on Experiment 1 (see Chapter 2). Briefly, the subject's right hand was positioned with the index finger resting on the pivoting plate (Figure 3.1) fixed to a potentiometer. Movement of the pivoting plate was mechanically limited to 20°. Surface EMG was recorded from the first dorsal interosseous (FDI) muscle using Ag/AgCL electrodes. The electrode was mounted on the dorsum of the middle phalange of the index finger. A black cardboard sheet over the subject's right hand prevented vision of their hand. A simple staircase procedure (Levitt, 1971) first established the shock intensity at which approximately 90% of stimuli delivered to the resting finger were detected. Intensity was varied by adjusting stimulator pulse-width. Occasionally, it was necessary to fine-tune the intensity level of the stimulus during a practice block. This intensity level then remained constant throughout the experimental blocks. The staircase procedure was repeated at the end of the experiment.

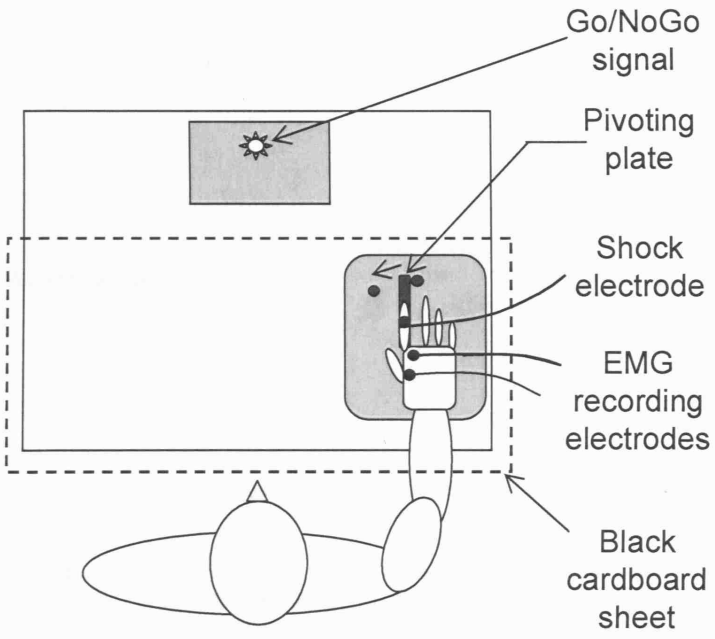

Figure 3.1. Experimental set-up. Subjects placed their right index finger on a pivoting plate. The Go/NoGo signal was presented via an LED in front of the subject. Direct vision of the hand was prevented.

In the *prior instruction* task, a verbal instruction "move" or "don't move" was given by the experimenter at the start of each trial (see Figure 3.2; Williams et al., 1998). After 1000 ms, a Go signal (a green LED for half the subjects and a red LED for the remainder) was presented for 700 ms. This acted as a response window encouraging fast responses, and thus allowing shock delivery in the critical interval for sensory suppression effects just prior to movement. The shock was delivered 50 ms before each subject's mean reaction time (the onset of EMG activity served as a measure of the reaction time in both experiments) from the practice block. Subjects made speeded right index finger abductions in response to the 'Go' signal on movement trials, but no response on non-movement trials.

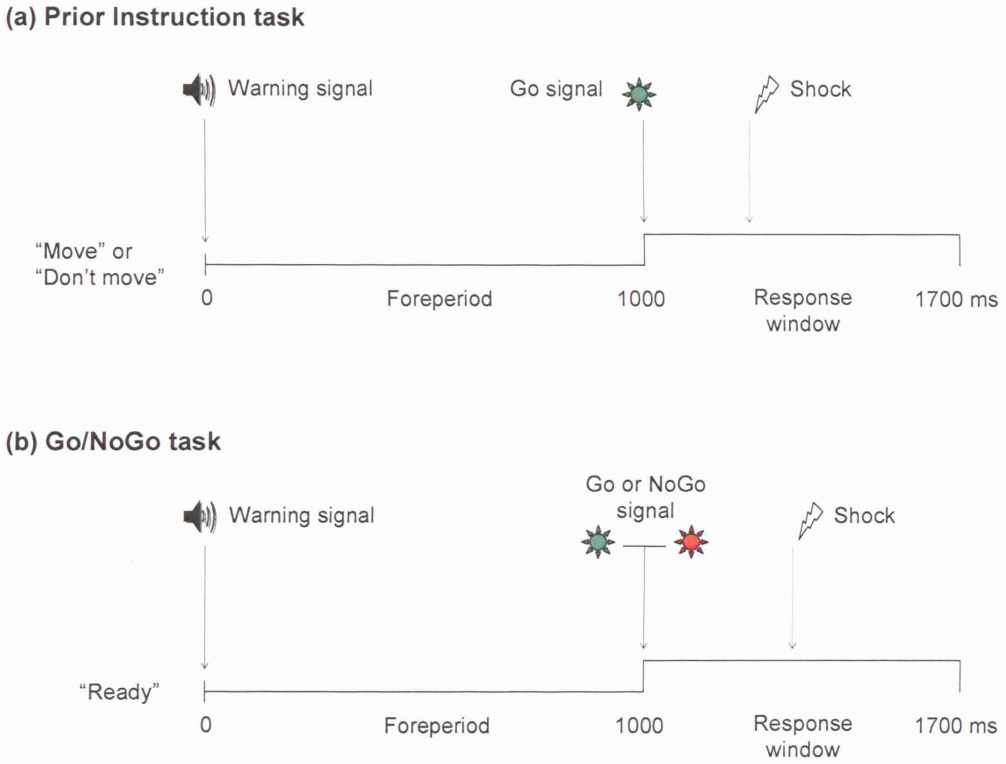

Figure 3.2. Experimental design. **(a)** In the *prior instruction* task, the experimenter gave a verbal instruction before the start of each trial, either 'move' or 'don't move'. An acoustic warning signal marked the start of the trial. After a delay of 1000 ms, the Go signal was presented. Subjects either moved or did not move their index finger during the 700 ms response window. **(b)** In the Go/NoGo task, the experimenter said 'ready' at the start of each trial. Subjects waited for and responded to the Go signal, but withheld movement following the NoGo signal. In both tasks, the shock was delivered 50 ms before each subject's mean reaction time.

In the Go/NoGo task, the start of each trial was announced by the experimenter saying "ready". The green LED 1000 ms later signalled 'Go' for 5 subjects and the red light signalled 'NoGo', while for the remaining subjects this order was reversed. After each trial, subjects reported verbally ('yes'/'no') whether they perceived a shock stimulus. No feedback was given. The experimenter then initiated the next trial after an intertrial interval of at least 1 second.

Subjects performed a practice block of 44 trials for both the Go/NoGo and *prior instruction* tasks. The order of the practice blocks was counterbalanced. Practice was followed by 4 experimental blocks of 44 trials. Each block consisted of 30 movement trials and 10 non-movement trials yielding a 3:1 Go to NoGo ratio. A further 4 catch trials with no shock stimulus were divided equally between Go and NoGo trials. The order of the blocks was interleaved for task i.e. ABAB or BABA, and the order of the trials was randomised.

3.4 Results

On catch trials lacking any shock stimulus only 0.63% false positive detections were recorded, indicating that subjects used a very conservative response strategy. A 2 x 2 ANOVA showed that there was no difference in the number of false positives on catch trials between tasks; $F(1,9)=1.000$; $p=0.343$ (see Table 3.2).

Table 3.2. The number of false positive catch trials pooled across subjects during movement and non-movement trials for the *prior instruction* and Go/NoGo tasks.

Trial Type	**Task**	
	Prior instruction	**Go/NoGo**
Movement	1	0
Non-movement	0	0

Errors of commission (i.e. when a non-movement trial was accompanied by EMG activity) were 3.0% and 5.5% for the *prior instruction* and Go/NoGo tasks respectively. Electromyographic activity was sometimes observed in the agonist FDI muscle in the absence of an overt finger movement. Errors of omission (i.e. movement trials without movement during the response window) occurred on 1.8% of movement trials. Both types of error trials were excluded when measuring the effects of sensory suppression.

3.4.1 Stability across time of perceptual performance at rest

The pre- and post-experiment staircases for the remaining 10 subjects showed similar shock intensity thresholds; mean pulse-widths were 20.8 and 19.7 μs respectively; $t(9)=1.819$; $p=0.102$.

3.4.2 Reaction times

Because sensory attenuation follows a precise time-course leading up to movement onset, the interval between shock and the onset of EMG activity must be similar across tasks if detection rates are to be compared. Reaction times for each subject were therefore trimmed to +/- 2 SD (excluding 3.8% of movement trials) and subjected to a one-way ANOVA. On 45% and 53% of movement trials in the *prior instruction* and Go/NoGo tasks respectively, the shock stimulus was delivered prior to the onset of EMG activity. The remaining trials were discarded because only sensory suppression of premotor origin was of interest here. Moreover, sensory suppression that occurs after EMG onset might be due to peripheral masking. The mean RTs for the Go/NoGo and *prior instruction* tasks were 331 ms and 230 ms respectively. This difference was significant $F(1,9)=76.417$; $p<0.0001$. However, the mean duration between shock and EMG activity did not differ significantly $F(1,9)=0.218$; $p=0.651$, suggesting that our adjustment of shock to each subject's mean RT in the practice block was successful.

3.4.3 Shock detection

Figure 3.3 shows shock detection rates in each task. A repeated measures ANOVA showed a significant main effect of movement $F(1,9)=141.211$; $p<0.0001$, no main effect of task $F(1,9)=3.261$; $p=0.104$, and a significant interaction between movement and task $F(1,9)=21.944$; $p=0.001$. Post-hoc t tests showed that this interaction arose because non-movement trials showed poorer detection in the Go/NoGo task than in the *prior instruction* task $t(9)=3.920$; $p=0.004$. Detection rates for movement trials were similar in both tasks $t(9)=0.761$; $p=0.466$.

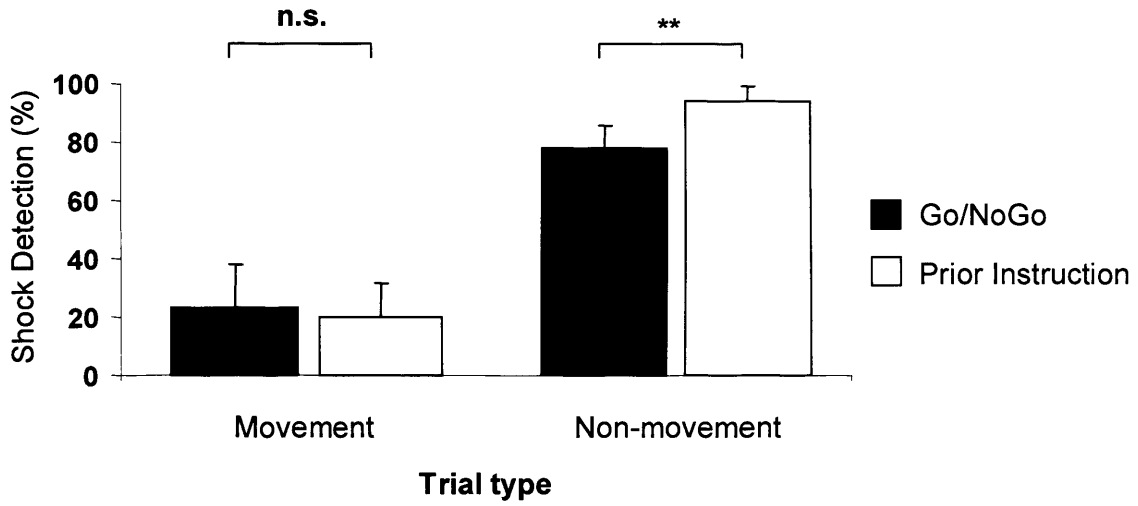

Figure 3.3. The mean percentage of stimuli detected (+/- SD) for movement and non-movement trials during the Go/NoGo and *prior instruction* tasks.

Table 3.3 shows the hypothesised levels of preparation for each trial type and the associated shock detection rates when the shock was delivered during the Go/NoGo and *prior instruction* tasks. Note that shock detection rates are similar for the movement trials in both tasks. However, for trials which did not involve any movement, shock detection rates were lower in the NoGo task (78%) than in *prior instruction* task (94%).

Table 3.3. Time-line of events showing the hypothesised levels of preparation and the actual shock detection rates for movement and non-movement trials for the *prior instruction* and Go/NoGo tasks.

Task	Trial	Movement probability	Motor Response	Hypothesised level of preparation	Shock detection (%)
Prior instruction	Movement	100%	Execute movement	Prepare	20
	Non-movement	0%	Do nothing	Do nothing	94
Go/NoGo	Go	75%	Execute movement	Prepare	23
	NoGo	75%	Inhibit movement	Prepare	78

3.4.4 Time-dependent changes in the detection of stimuli applied to the moving digit

To study the time-course of any variation in performance during movement, the data were pooled from all subjects, trials were grouped into 50 ms bins relative to EMG onset and the graph plotted (Figure 3.4). However, it should be noted that not all subjects necessarily contribute to each data point, particularly near the edges of the graph. While pooled data gives the overall picture, statistical analyses for sensory suppression effects were only carried out as repeated measures.

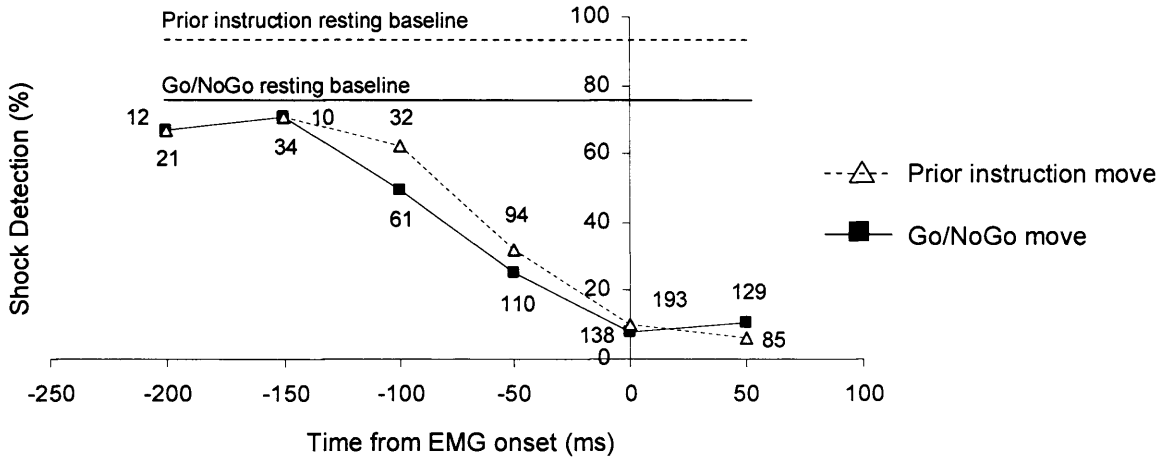

Figure 3.4. Effects of index finger movement on the detection of fixed intensity stimuli applied to the moving and non-moving finger during the Go/NoGo and *prior instruction* tasks. Detection performance over time is plotted relative to the onset of EMG (0 ms); a negative number indicates that the shock precedes EMG onset. The values next to the data points refer to the number of trials represented at that data point, pooled over subjects.

Inspection of Figure 3.4 reveals the time-dependent curve in shock detection rates, indicating premovement sensory suppression for both tasks consistent with Experiment 1 and Williams et al. (1998). Furthermore, linear regression equations indicated time-dependent changes in performance with slopes significantly different from zero for the *prior instruction* ($p=0.005$) and the Go/NoGo ($p=0.004$) tasks confirming a time-dependent change in performance for both tasks. The proportion of stimuli perceived was calculated for each bin. A 2 x 6 repeated measures ANOVA was carried out on shock detection rates for the factors task (Go/NoGo vs. *prior instruction*) and time-bin (200, 150, 100, 50, 0 and post-50 ms relative to EMG onset). There was no significant main effect of task $F(1,9)=0.005$; $p=0.945$. There was a significant main effect of time-bin $F(5,45)=17.899$; $p<0.0001$. However, the interaction task by time-bin was not significant $F(5,45)=0.231$; $p=0.897$.

3.5 Discussion

Here, we have compared sensory attenuation on movement and non-movement trials to investigate processes of movement preparation and cancellation. We found that detection rates on movement trials were lower than on non-movement trials, consistent with sensory suppression (Experiment 1; Williams et al., 1998). Furthermore, we compared sensory suppression effects on a Go/NoGo task with the *prior instruction* task. In the *prior instruction* task, each trial was preceded by a verbal instruction (to move or not to move). There was no difference between tasks in detection for movement trials. However, detection rates for non-movement trials were significantly lower in the NoGo than in the *prior instruction* task, suggesting an element of sensory suppression associated with actions which are prepared, but then inhibited before execution. Peripheral feedback cannot have played a role in this attenuation, since no actual movement occurred. Previous studies using transcranial magnetic stimulation (TMS) have also provided evidence for a premotor contribution to sensory suppression (e.g. Voss et al., 2006; Haggard and Whitford, 2004). However, while those studies have provided *neural* evidence for a cognitive component to sensory suppression, the current study in contrast, provides *behavioural* evidence using sensory variables.

In this study, the prior instruction provided subjects with complete information about the metrics of the impending task as reflected by the shorter RT relative to the Go/NoGo task. The high degree of response certainty permitted unambiguous selection of the response and full specification of its parameters. The finding that there was no significant difference in shock detection levels on movement trials for both the *prior instruction* and Go/NoGo tasks hints at a motor preparation process that activates to approximately the same extent even when the task demands are different. Movement-related sensory suppression processes were relatively invariant with subjects establishing a high level of motor preparation during both tasks. Alternatively, on non-movement trials a substantial difference between the two tasks is the amount of inhibitory activity required to stop or cancel any intention to move. Crucially, in the *prior instruction* task on non-movement trials, subjects knew in advance that no action was required; therefore there was no need

to prepare for an action. In contrast, in the Go/NoGo task, subjects could not anticipate the response, as the visual signal in each trial provided that information only at the end of the foreperiod. Therefore, subjects were required to discriminate the signal and then decide whether to move or not. After a NoGo signal, they must cancel the prepared response. This cancellation activity on NoGo trials seems to have resulted in significantly lower shock detection rates than on non-movement trials in the *prior instruction* task where no preparation or movement was required.

A methodological issue in the current experiment is that both tasks required different stimulus discriminations. In the *prior instruction* task, a green test signal was used on every trial, in-line with the original procedure (Williams et al., 1998), whereas the Go/NoGo task utilised a Go and a NoGo signal of different colours (e.g. green = Go and red = NoGo). It is therefore possible that a part of the difference in shock detection rates observed during non-movement trials might reflect purely perceptual processing demands. However, the attentional literature would suggest that the additional information provided by varying stimulus colour would, if anything, have an arousing effect (e.g. Stein and Meredith, 1993). This would then facilitate shock detection on NoGo trials, a prediction opposite to the current result.

Here, we provide evidence that preparation is strongly linked to premovement sensory suppression. There was no difference in shock detection rates during movement trials between different instruction conditions. Motor processes across both tasks were relatively invariant. However, on NoGo trials when a prepared movement was successfully cancelled, subjects detected significantly fewer shock stimuli than in non-movement trials of a *prior instruction* task when they presumably neither prepared actions in advance, nor inhibited them. Using sensory variables, differences in the amount of preparation were detected even in the absence of overt behaviour. Sensory suppression can occur due to the preparation of actions, even when they are not executed. Here, we have measured sensory suppression effects that occurred after the signal. However, preparation processes are presumed to also take place during the foreperiod of a

trial (Näätänen and Niemi, 1981). The aim of the next experiment is to examine possible foreperiod preparation effects using sensory suppression.

Chapter 4: Sensory suppression during a preparatory foreperiod

4.1 Abstract

The current design aimed to partly replicate Experiment 2, again comparing sensory suppression during a Go/NoGo and a *prior instruction* task. We replicated the findings of Experiment 2. Thus, subjects detected less shocks just prior to movement than when doing nothing. The Go/NoGo effects of Experiment 2 were also replicated. However, here the focus was on whether preparatory processes during the foreperiod of a trial cause sensory suppression. When the shock was delivered during the foreperiod, just *prior* to the Go/NoGo signal, sensory suppression was the same for both Go and NoGo trials. This suggests that subjects were equally prepared on these trials. In contrast, in the *prior instruction* task shock detection was only suppressed on movement trials confirming that subjects did not prepare when the instruction was not to move. These results support the idea that sensory suppression can begin in advance of trigger stimulus onset, if the response is known in advance. It is concluded that sensory suppression has a strong cognitive component.

4.2 Introduction

"What takes place during the foreperiod?" Näätänen and Niemi posed the question in the final sentence of their 1981 article. In the present experiment, we will deliver shocks during the foreperiod to investigate the cognitive processes of preparation in terms of their effects on sensation and thus answer the above question. In a typical RT paradigm, a trial consists of the warning signal, the foreperiod, the Go (or NoGo) stimulus and the response window (see Figure 4.1).

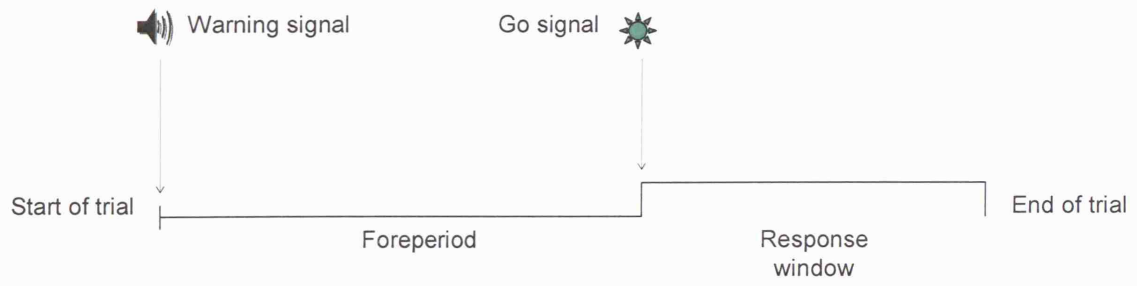

Figure 4.1. Typical trial design. The trial consists of a warning signal which marks the start of the trial, a foreperiod or preparation interval, a signal and a response window where the required response is carried out.

Preparatory processes are usually presumed to take place during the foreperiod of a trial (Näätänen and Niemi, 1981). Näätänen and Merisalo (1977) defined preparation by suggesting that it consists of "carrying out everything of a response that can be carried out in advance". Anticipatory behaviour starts with the presentation of a warning stimulus, indicating to subjects that after an amount of time, a stimulus will be presented which can indicate to subjects whether to make a movement or not. Preparation should facilitate the correct perception and the right response execution during a task. A correct perception carries with it a selection of the relevant information and a suppression of available irrelevant information. Efficient response execution implies selection and excitation of the relevant peripheral motor structures and a suppression of activity in irrelevant peripheral motor structures (Brunia and Böcker, 1995). Preparation leads to more efficient behaviour, resulting in a shorter reaction time.

Preparation is manifest in a number of physiological response systems. Heart-rate shows the well-known pattern of an early deceleration and a terminal deceleration prior to the arrival of the response signal and the execution of the response (Lacey and Lacey, 1970). Changes in excitability of spinal motoneurons as a manifestation of preparation can be studied by means of spinal reflexes evoked during the foreperiod of an RT task (Hasbroucq et al., 1997). Preparation can also be investigated by recording slow potentials, reflecting changes in excitability of cortical neurons during the foreperiod of a RT task. At the cortical level it implies activation of motor areas, setting the spinal and brainstem motor structures ready for action. Crucially for our purposes, preparation also implies activation of the somatosensory areas to prevent irrelevant input from disturbing the correct response execution and activation of sensory areas in the modality of the Go or NoGo signal (Brunia and Böcker, 1995).

One model that is useful for describing effects of preparation on the motor system was proposed by Näätänen (1970; see also Niemi and Näätänen, 1981). The basic concept of Näätänen's model is 'motor readiness', which is conceived as a delicate balance between excitatory and inhibitory motor commands (see Figure 4.2).

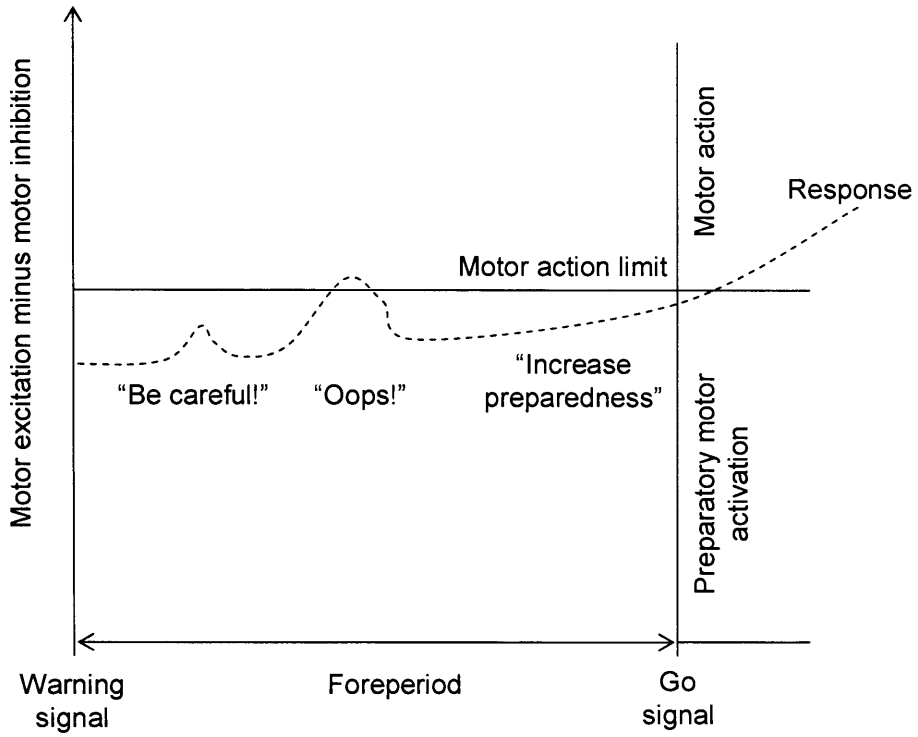

Figure 4.2. An imagined course of motor readiness (dashed line) during a fixed foreperiod task. If the level of motor readiness rises above the motor action limit a response is inevitable. At the start of the foreperiod, the subject maintains relatively low levels of preparation as maintaining readiness is costly and aversive. Towards the end of the foreperiod, the subject increases their level of preparation towards the motor action limit. A premature response would occur if the level of preparation was increased beyond the motor action limit during the foreperiod (adapted from Näätänen, 1970 and Mattes et al., 1997).

During task performance, the subject establishes a criterion known as the motor-action limit, and triggers an overt response when motor readiness reaches this criterion. The model proposes that subjects adjust their motor readiness closer to the motor limit only when they expect the response signal to occur as maintaining a high level of preparation is energy-consuming and aversive (Niemi and Näätänen, 1981). Control over the readiness level is assumed to be imprecise. Even in conditions where the foreperiod is fixed, readiness to respond can still vary, owing to central correcting commands to maintain the desired level of motor preparation. According to the model, subjects have no direct way of monitoring the momentary distance between the actual degree of motor preparation and the motor action limit. Thus, maintaining a high degree of motor

preparation to be able to respond fast when the stimulus is delivered requires continuous correction via excitatory and inhibitory mechanisms. Therefore motor preparation fluctuates, sometimes being very near the motor action limit, and sometimes being drawn back to a safer level. If it 'flows over', the result is a premature response (as illustrated by "oops!" in Figure 4.2).

Here, the focus was on whether preparatory processes during the foreperiod of a trial cause sensory suppression. Centrally-mediated sensory suppression i.e. attenuation that precedes EMG onset, is thought to be related to the preparation of movements (Experiment 2; also Voss et al., 2006). We might reasonably expect levels of preparation to be equal on both Go and NoGo trials during the foreperiod of the Go/NoGo task as subjects must await the signal that informs them whether to move or not. As the Go signal is prepotent, subjects will tend to prepare to move on every trial. Likewise, in the *prior instruction* task, subjects need only prepare a movement when verbally instructed to do so before the start of the trial. In contrast, on non-movement trials, subjects do not need to prepare, they simply have to do nothing (Table 4.1).

Table 4.1. Time-line of events for the *prior instruction* and Go/NoGo tasks when the shock is delivered before the signal during the foreperiod. In the *prior instruction* task subjects, having received a verbal instruction before the start of the trial, are predicted to prepare strongly on movement trials and not to prepare on non-movement trials. Alternatively, in the Go/NoGo task, subjects are predicted to prepare strongly during the foreperiod on all trials as the prepotent response is Go.

Task	Trial	Verbal instruction	Motor response	Hypothesised level of foreperiod preparation
Prior instruction	Movement	"Move"	Execute movement	Prepare
	Non-movement	"Don't move"	Do nothing	Do nothing
Go/NoGo	Go	None	Execute movement	Prepare
	NoGo	None	Inhibit movement	Prepare

When the shock is delivered *after* the signal, the current design aims to replicate the last experiment demonstrating premotor sensory suppression of movement i.e. less shocks detected prior to movement than during non-movement. We additionally extend the previous design by delivering shocks at the end of the foreperiod and prior to the Go/NoGo signal, in order to investigate preparatory processes during this interval. In the *prior instruction* task, we predict shock detection to be greater on non-movement trials, where subjects do not need to prepare a movement, than on movement trials, where subjects prepare to move. In the Go/NoGo task, we predict no difference in shock detection rates between Go and NoGo trials, since subjects are predicted to prepare to move on every trial.

4.3 Materials and Methods

4.3.1 Subjects

Fifteen paid subjects took part with local ethical committee permission. The data from 4 subjects were excluded because their detection of cutaneous shocks at rest was unstable across the experiment (post-test detection varied more than +/-15% from pre-experiment levels). Data from the remaining 11 subjects (7 female, 10 right-handed, mean age 24.2 (SD = 6.7) years were included in the final analysis.

4.3.2 Procedure

The set-up and procedures were identical to Experiment 2, with the following exceptions. The shock was delivered randomly either 50 ms before each subject's mean response time from the practice block *or* 50 ms before the Go or NoGo signal (see Figure 4.3).

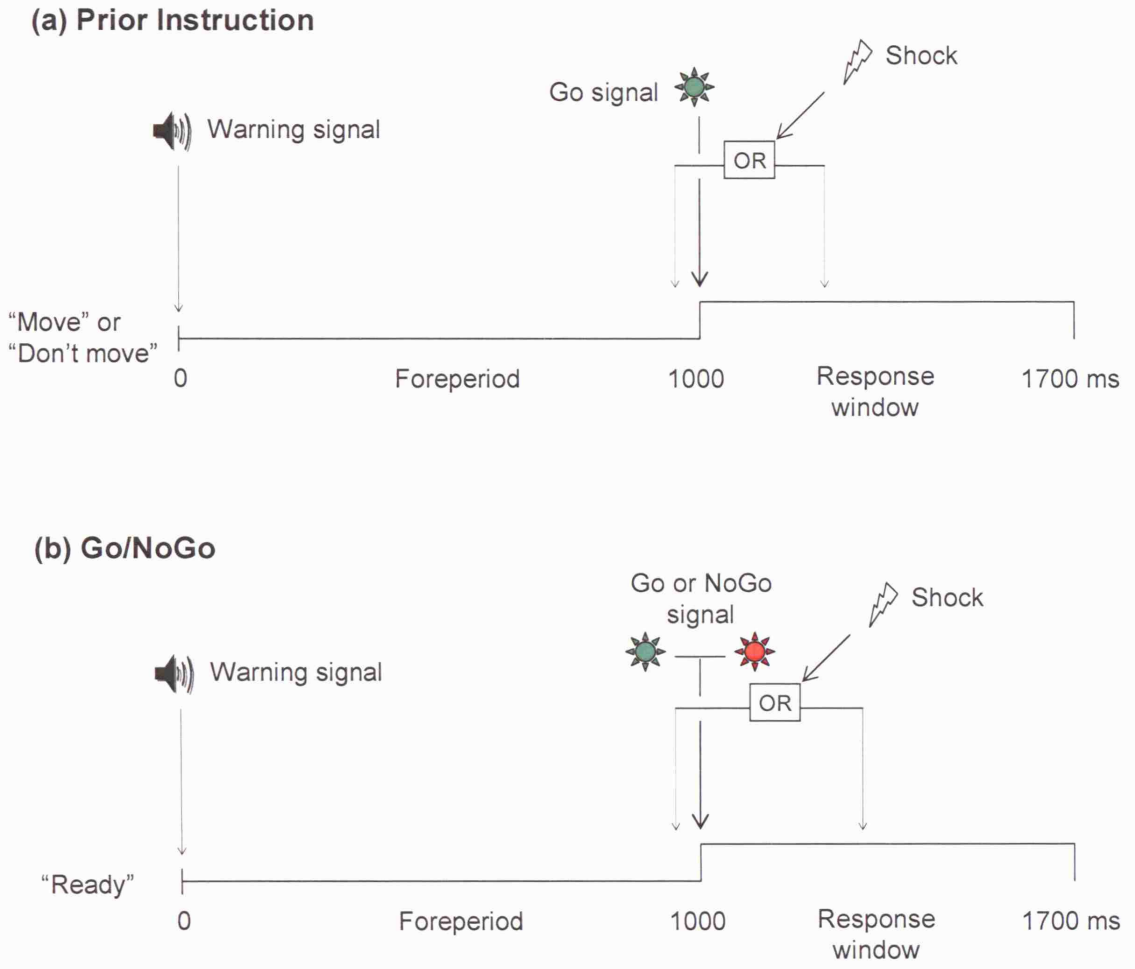

Figure 4.3. Experimental design. **(a)** In the *prior instruction* task, the experimenter gave an instruction before the start of each trial, either 'move' or 'don't move'. An acoustic warning signal marked the start of each trial. The Go signal was presented after 1000 ms. Subjects either moved or did not move their index finger during the 700 ms response window. **(b)** In the Go/NoGo task, the experimenter said 'ready' at the start of each trial. Subjects waited for and responded to the Go signal, but withheld movement following the NoGo signal. In both tasks, the shock was delivered either 50 ms prior to the onset of the signal or 50 ms before each subject's mean reaction time (RT).

Subjects performed a practice block of 44 trials, followed by 6 experimental blocks of 54 trials, 3 for the *prior instruction* and 3 for the Go/NoGo tasks. Each block consisted of 36 movement trials and 12 non-movement trials. On half of these trials the shock was delivered 50 ms before the imperative signal while on the remaining half, the shock was delivered 50 ms prior to the subject's mean reaction time. A further 6 catch trials (no shock stimulus) were divided equally between movement and non-movement trials. The

order of the blocks was interleaved for task i.e. ABABAB or BABABA, and the order of the trials was randomised.

4.4 Results

On catch trials only 0.8% false positive detections were recorded, indicating that subjects used a conservative response strategy. There was no difference in the number of false positives on catch trials between tasks; $t(10)=1.000$; $p=0.341$ (see Table 4.2).

Table 4.2. The number of false positive catch trials pooled across subjects during movement and non-movement trials for the *prior instruction* and Go/NoGo tasks.

Trial Type	**Task**	
	Prior instruction	**Go/NoGo**
Movement	0	0
Non-movement	1	2

Errors of commission (i.e. when a non-movement trial was accompanied by EMG activity) were 3.0% and 5.8% for the *prior instruction* and Go/NoGo tasks respectively. A 2 x 2 repeated measures ANOVA was performed on the rate of errors of commission for the factors task (*prior instruction* vs. Go/NoGo); and shock-timing (50 ms before signal or 50 ms before mean individual RT). There was no significant main effect of movement $F(1,10)=2.750$; $p=0.137$ and no main effect of task $F(1,10)=2.872$; $p=0.121$. There was also no interaction $F(1,10)=2.189$; $p=0.170$. The finding that there was no difference in the number of errors of commission when the shock was delivered before or after the signal suggests that the presence of the shock did not affect movement preparation. Errors of omission (i.e. movement trials without movement during the

response window) occurred on 1.8% of movement trials. Error trials were excluded when measuring the effects of sensory suppression.

4.4.1 Stability across time of perceptual performance at rest

The pre- and post-experiment staircases showed similar shock intensity thresholds in the remaining subjects (mean pulse-widths=25.0 and 22.9 μs respectively: $t(10)=1.145$; $p=0.279$).

4.4.2 Reaction times

Because sensory attenuation follows a precise time-course leading up to movement onset, the interval between shock and the onset of EMG activity must be similar across tasks if detection rates are to be compared. RTs for each subject were trimmed to +/- 2 SD (excluding 5.8% of movement trials) and subjected to a one-way ANOVA. The mean RTs for the Go/NoGo and *prior instruction* tasks were 316 ms (SD = 50 ms) and 231 ms (SD = 49 ms) respectively. This difference was significant $t(10)=10.367$; $p<0.0001$ (two-tailed). However, the mean duration between shock and EMG activity did not differ significantly $t(10)=1.566$; $p=0.148$ (two-tailed), suggesting that our adjustment of shock to each subject's mean RT in the practice block was successful. On 88.4% of movement trials, the shock was delivered prior to EMG onset. For the remaining 11.6% of trials, the shock followed EMG onset. These trials were excluded from the sensory suppression analysis.

4.4.3 Shock detection

Figure 4.4(a) and (b) shows shock detection rates in the *prior instruction* and Go/NoGo tasks and for each of the shock intervals (pre-signal or post-signal). A 2 x 2 x 2 repeated

measures ANOVA for the factors task (*prior instruction* vs. Go/NoGo); movement (movement vs. non-movement trials) and shock-timing (50 ms before signal or 50 ms before mean individual RT) was carried out. There was a significant main effect of movement $F(1,10)=144.484$; $p<0.0001$ and a main effect of task $F(1,10)=16.297$; $p=0.002$. There was no main effect of shock-timing $F(1,10)=0.294$; $p=0.600$.

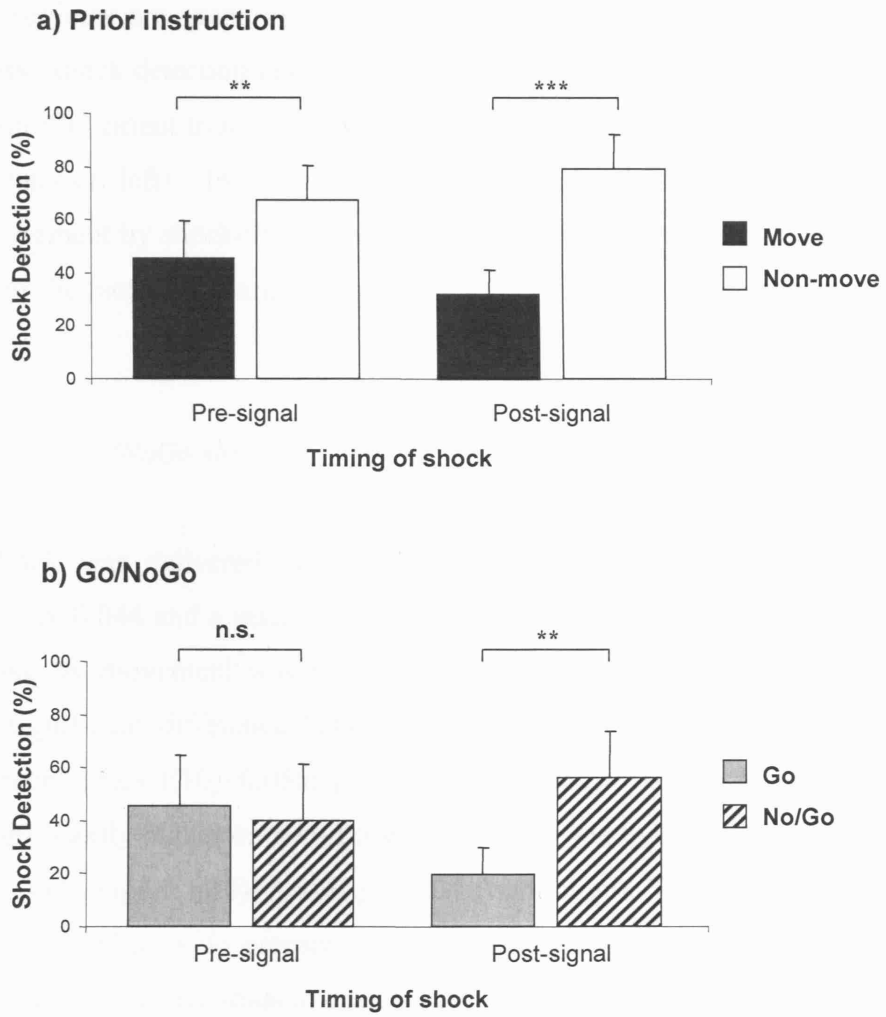

Figure 4.4. The mean percentage of shocks detected (+/- SD) for (a) the *prior instruction* and (b) Go/NoGo tasks for shocks delivered at the end of the foreperiod (pre-signal) or 50 ms before mean reaction time after the Go/NoGo signal (post-signal).

There was a significant task by movement interaction $F(1,10)=13.171$; $p=0.005$. The interaction movement by shock-timing was also significant $F(1,10)=36.909$; $p<0.0001$. However, the task by shock-timing interaction was not significant $F(1,10)=0.261$; $p=0.621$. The 3-way interaction task by movement by shock-timing also reached significance $F(1,10)=5.820$; $p=0.037$. There was no difference in shock detection rates on the Go/NoGo task when the shock was delivered during the foreperiod suggesting equal amounts of preparation (Figure 4.4; bottom panel, 2 bars on left). This is consistent with the fact that subjects are preparing to move on every trial. In contrast, in the *prior instruction* task, shock detection rates during the foreperiod on non-movement trials were greater than on movement trials thereby demonstrating sensory suppression (Figure 4.4; top panel, 2 bars on left). In order to separately examine events before and after the signal, the movement by shock-timing interaction was followed up with two separate 2 x 2 ANOVAs for the factors task and movement.

4.4.3.1 Before the Go/NoGo signal

When the shock was delivered before the signal, there was a main effect of task $F(1,10)=5.293$; $p=0.044$ and a main effect of movement $F(1,10)=111.175$; $p=0.017$. The interaction task by movement was also significant $F(1,10)=17.805$; $p=0.002$. T tests revealed no significant difference between the Go/NoGo and *prior instruction* tasks during movement trials $t(10)=0.056$; $p=0.956$ (two-tailed). However, shock detection rates were significantly higher on non-movement trials when there was a *prior instruction* relative to a NoGo signal $t(10)=3.654$; $p=0.004$ (two-tailed). This finding is consistent with the view that subjects do prepare actions in advance of a Go/NoGo signal, and do not prepare in non-movement trials of a *prior instruction* task.

A 3 x 1 ANOVA carried out to compare detection rates when the shock was delivered before the signal for movement trials in the *prior instruction* task and the Go and NoGo trials in the Go/NoGo task revealed no significant difference between the three trial types; $F(2,20)=0.627$; $p=0.496$ (compare Figure 4.4(a) black bar on left with (b) 2 bars on left).

This suggests that subjects were as equally prepared during the foreperiod of the Go/NoGo task as in the foreperiod of the *prior instruction* task, when they knew with certainty to move once the signal was presented.

Table 4.3 shows the hypothesised levels of preparation for each trial type and the associated shock detection rates when the shock was delivered during the foreperiod of a trial. Note that shock detection rates were similar 45%, 46% and 40%, when it was predicted that subjects would prepare strongly on *prior instruction* movement, Go and NoGo trials respectively.

Table 4.3. Time-line of events showing the hypothesised level of preparation and the actual shock detection rates when the shock is delivered during the foreperiod for movement and non-movement trials in the *prior instruction* and Go/NoGo tasks.

Task	Trial	Motor response	Hypothesised level of foreperiod preparation	Shock detection rates (%)
Prior instruction	Movement	Execute movement	Prepare	45
	Non-movement	Do nothing	Do nothing	68
Go/NoGo	Go	Execute movement	Prepare	46
	NoGo	Inhibit movement	Prepare	40

4.4.3.2 After the Go/NoGo signal

When the shock was delivered after the signal, there was a main effect of task $F(1,10)=14.894$; $p=0.003$ with shock detection performance during the *prior instruction* task being greater than during the Go/NoGo task. There was also a main effect of movement $F(1,10)=111.175$; $p<0.0001$ with differences in shock detection between

movement and non-movement trials of 47% for the *prior instruction* task and 36% for the Go/NoGo task. However, the interaction task by movement was not significant $F(1,10)=2.911$; $p=0.119$.

4.4.4 Time-dependent changes in the detection of stimuli applied to the moving digit

In order to study the critical interval of sensory suppression just prior to onset of EMG, all movement trials, both pre-signal and post-signal were displayed in a response-locked way. These trials were grouped into 50 ms bins relative to EMG onset and the proportion of stimuli perceived was calculated for each bin. Due to natural trial to trial variation in RTs, this yielded five bins when shocks were delivered prior to the signal and 6 bins when shocks were delivered after the signal (see Figure 4.5).

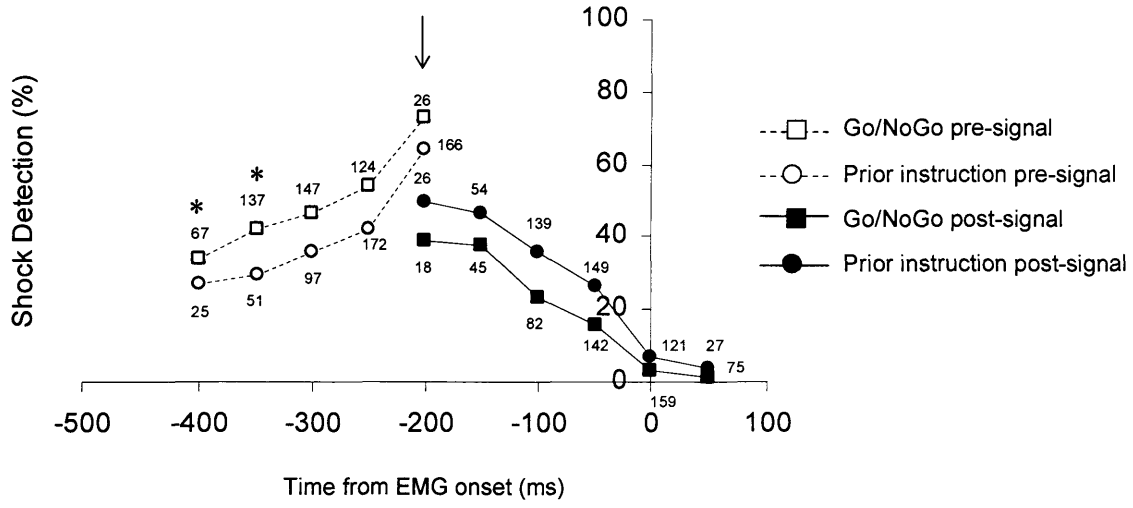

Figure 4.5. Effects of right finger abduction on the detection of stimuli applied to the moving finger for the Go/NoGo and *prior instruction* tasks for shocks delivered 50 ms before the Go signal (pre-signal) or 50 ms prior to each subject's mean reaction time after the Go signal (post-signal). All data are response-locked, whether shocks were delivered before or after the Go signal. The arrow denotes the overlap (see Results section for details). Data were sorted into 50 ms bins. The values next to each data point refer to the number of trials represented at that data point. An asterisk denotes a significant difference in shock detection performance ($p<0.05$; one-tailed) between both tasks for that time-bin (see also Table 4.4).

The upward gradient of the slopes for the pre-signal curves was not predicted (see dashed lines; Figure 4.5). Detection of shock stimuli should diminish with increasing proximity to EMG rather than increase. Also shock detection was greater on movement trials pre-signal in the Go/NoGo task than the *prior instruction* task. However, this tendency in shock detection for the two tasks was reversed, post-signal (see Discussion for commentary).

The time-bin 200 ms prior to EMG onset (see arrow, Figure 4.5) is interesting because it represents an overlap in the data points. The shock was always delivered 200 ms before EMG onset for all trials represented at this point yet in some of these trials the visual Go signal had already occurred, while in others it had not. That is, pre-signal and post-signal trials could be compared for this bin, allowing us to isolate the effects of the signal per se on sensory suppression, while keeping the relation to movement constant. The difference in mean pre-signal and post-signal shock detection rates for time-bin 200 ms prior to EMG onset was 24%. However, a post-hoc t test revealed no differences between pre- and post-shock detection rates for the *prior instruction* task $t(10)=1.543$; $p=0.154$ nor for the Go/NoGo task $t(10)=0.699$; $p=0.501$, due to high inter-subject variability. This analysis suggests that sensory suppression can produce as much sensory suppression before a Go signal as after it, once differences in response-locked timing between shock and EMG onset are controlled for. This is consistent with a unitary process of sensory suppression that is perhaps related to Näätänen's (1970) unitary motor inhibition level concept.

4.4.5 Shocks delivered prior to the signal

A 2 x 5 repeated measures ANOVA carried out on shock detection rates for the factors task (*prior instruction* vs. Go/NoGo) and time-bin (400, 350, 300, 250 and 200 ms before the signal) did not yield a main effect of task $F(1,10)=1.551$; $p=0.241$. The main effect of time-bin was significant $F(4,40)=4.334$; $p=0.030$ as was the interaction $F(4,40)=3.378$; $p=0.042$. Follow-up t tests (one-tailed) were performed and these results are presented in

Table 4.4. Significant differences were found for the two earliest time-bins relative to EMG onset.

Table 4.4. Follow-up t tests (p values are one-tailed) comparing both tasks at each time-bin when the shock was delivered before the signal. Time-bins are relative to onset of EMG (0 ms).

Time-bin (ms)	p value (see also Figure 4.5)
400	0.012
350	0.037
300	0.327
250	0.472
200	0.177

4.4.6 Bins when the shock was delivered after the Go signal

A 2 x 6 repeated measures ANOVA was carried out on shock detection rates for the factors task (*prior instruction* vs. Go/NoGo) and time-bin (200, 150, 100, 50, 0 ms relative to EMG onset and 50 ms after EMG onset). There was no main effect of task $F(1,10)=1.551$; $p=0.314$. The main effect of time-bin was significant $F(5,50)=10.122$; $p<0.0001$. The interaction failed to reach significance $F(5,50)=1.333$; $p=0.283$.

4.5 Discussion

In the current experiment, we delivered shocks before and after the signal. For shocks delivered after the signal, we replicated the findings of Experiment 2. Subjects detected fewer shocks when moving than when not moving. There was no difference between tasks in detection for movement trials. Subjects detected fewer shocks in the NoGo than in *prior instruction* non-movement trials when no overt behaviour was involved, replicating the finding that an element of sensory suppression remains when a prepared

action is cancelled. Extending these findings, we also delivered shocks at the end of the foreperiod to investigate if there were differences in levels of preparation between the *prior instruction* and Go/NoGo tasks before the signal. In the foreperiod of a Go/NoGo task, subjects should be prepared to move during both Go and NoGo trials because of the 3:1 prepotency of the Go signal. Likewise, in the foreperiod of a *prior instruction* task, subjects should be highly prepared when the verbal pre-instruction is to move. In stark contrast, when the pre-instruction is not to move then subjects need not prepare. If preparation is strongly linked to sensory suppression then these levels of preparation should be reflected in the shock detection rates. That is exactly what was observed (see Results; Table 4.3). When subjects were predicted to prepare strongly, shock detection rates were low (45, 46 and 40% on *prior instruction* movement, Go and NoGo trials respectively). However, when subjects did not prepare a movement and could presumably allocate more resources to the perception of the shock stimulus, then detection rates were significantly greater (68%). We conclude that preparation is strongly linked to sensory suppression. When subjects are highly prepared, they feel less than when they do not need to prepare. Here, we have extended the original findings on sensory suppression (e.g. Williams et al., 1998) to the foreperiod of a trial.

Recent studies (Chapman and Beauchamp, 2006) have suggested a possible "backward masking" explanation of premovement sensory suppression effects during abduction of a digit. Backward masking refers to the decrease in sensation that occurs when a second input occurs soon after an earlier stimulus. It was reported that the time-course for modulation of tactile detection by active and passive abduction of the index finger can be identical (Chapman and Beauchamp, 2006; Williams and Chapman, 2002) with detection declining before movement onset in both cases. This is surprising as shock detection rates for passive movements should not decline *prior* to movement (see Chapter 1). The timing of attenuation for passive movements should at least coincide with the onset of electromyographic muscle activation.

However, studies of backward masking in the somatosensory system suggest that both inhibition and disinhibition of sensory cortex occur too rapidly to explain the latency

difference in our data. Thus, Laskin and Spencer (1979) found that the masking effect of a second stimulus on a first was abolished if an interval of 50 ms or more was inserted between the two stimuli, suggesting relatively rapid switching of sensory gains. In the present study, the mean interval between the shock stimulus and the onset of EMG activity was in excess of 200 ms for both tasks (see Figure 4.3). The finding that sensory suppression was observed in the preparatory foreperiod of a trial, well in advance of muscle activation and movement onset, cannot easily be explained by backward masking (Chapman and Beauchamp, 2006). This is strong evidence of a cognitive component to sensory suppression and argues against backward masking accounts of premovement sensory suppression (Chapman and Beauchamp, 2006).

The basic concept of Näätänen's model (Näätänen, 1970; Niemi and Näätänen, 1981), as described in the Introduction, is that motor preparation is conceived as a delicate balance between excitatory and inhibitory motor commands (see Figure 4.2). Control over motor preparation is assumed to be imprecise and requires continuous correction via excitatory and inhibitory mechanisms. Therefore, according to the model, motor preparation fluctuates erratically, sometimes being very near the motor action limit, and sometimes being drawn back to a safer level. Our data show that there was no difference in shock detection rates when the probability of movement was 100% (*prior instruction* task) or 75% (Go/NoGo task) suggesting that levels of motor preparation during the foreperiod were similar in both instances. This finding suggests an invariance in motor preparation processes and is somewhat at odds with the delicate balance between excitation and inhibition described in Näätänen's (1970) model. It is possible that sensory suppression acts to keep the motor system in check in order to prevent the occurrence of a premature response.

Our results revealed an unexpected finding. The upward gradient of the slopes for the pre-signal curves was not predicted (see dashed lines; Figure 4.5). Detection of shock stimuli should diminish with increasing proximity to EMG rather than increase. At first glance, this finding seems odd; however, caution is required when interpreting these data. The time certainty of the foreperiod - fixed at one second - may have been accompanied

by an increase in arousal (Niemi and Näätänen, 1981). Trials in the time-bin 200 ms before EMG onset (as indicated by the arrow in Figure 4.5) have faster reaction times than trials in the time-bin 400 ms before EMG onset. Faster reaction times may be an indication that subjects were more attentive when performing these trials. Higher levels of attention would then also explain why subjects detected more shocks on these trials. It is known that attending to a task can improve task performance (e.g. Meredith and Stein, 1993). A common mechanism of general arousal, equally affecting shock detection and RT performance may explain the data. Further inspection of Figure 4.5 reveals that more shocks were detected on movement trials pre-signal in the Go/NoGo task than the *prior instruction* task. Interestingly, this tendency in shock detection for the two tasks was reversed post-signal. This tendency might simply reflect the greater attentional demand of monitoring two signals (the Go and NoGo signal) in the Go/NoGo task rather than one signal (Go signal only) in the *prior instruction* task. While these effects are interesting, clearly more research is required before any definite conclusions can be drawn.

A methodological issue in Experiment 2 and the current experiment is that both tasks required different stimulus discriminations. In the *prior instruction* task, a green test signal was used on every trial (Williams et al., 1998), whereas the Go/NoGo task utilised a Go and a NoGo signal of different colours (e.g. green = Go and red = NoGo). One possibility was that the difference in sensory suppression effects between the two tasks might reflect purely perceptual processing demands. Previously, Experiment 2 could not rule out this possibility. Here, we provide evidence that this was not the case as differing sensory suppression effects were also observed *before* the signal during both tasks. Variations in sensory suppression therefore seem to reflect the differing cognitive demands of both tasks.

Many cognitive models of motor control rest on the assumption that subjects are more prepared following a prior instruction and that important aspects of motor preparation can begin in advance of response stimulus onset when the response is known in advance (e.g. DeJong et al., 1990; Miller and Low, 2001; Smid et al., 2000). Here we provide evidence for a strong cognitive component to sensory suppression, replicating Experiment

2 and linking sensory suppression to preparation. We also extend the original findings on sensory suppression (e.g. Williams et al., 1998) to the foreperiod of a trial. Sensory suppression can occur even before the Go signal triggering execution processes is presented. These findings suggest that the signal is not responsible for sensory suppression effects. Furthermore, our results argue against backward masking explanations of movement-related sensory suppression in the distal digit. Taken together, the findings provide evidence for premotor and pre-signal sensory suppression and confirm that the S-D (sensory-detection) task provides a useful tool for exploring "what takes place during the foreperiod?" (Näätänen and Niemi, 1981).

Experiments 2 and 3 have shown that when a prepared movement is cancelled, the sensory system is suppressed. The aim of the next experiment is to measure how long it takes for the sensory and motor systems to recover from sensory suppression after the successful cancellation of a movement.

Chapter 5: Sensory suppression and the internal structure of stopping

5.1 Abstract

In a Go/NoGo task, subjects prepared a response, and then either executed it in response to a subsequent Go signal, or cancelled the movement if a NoGo signal occurred. The delay between the NoGo signal and shock was varied (cf., Experiments 2 and 3). Detection rates improved monotonically as the interval increased from 0 up to 200 ms. The recovery from sensory suppression offers a new way of measuring the processes triggered by a NoGo signal. Our results suggest that when a prepared movement is inhibited the dismantling of the sensory consequences of the motor command takes at least 200 ms. The rate of development of sensory suppression on Go trials proceeded more quickly than the rate of recovery from suppression on NoGo trials. The result is discussed in the context of a premotor origin of sensory suppression and as evidence about the time-course of the mechanisms underlying sensory suppression.

5.2 Introduction

Withholding a prepared action is a critical aspect of voluntary control (De Jong et al., 1995). Two closely-related paradigms, the stop-signal and the Go/NoGo paradigms, are commonly used to explore inhibitory control. In the latter, subjects generate a motor response upon presentation of a Go signal, and attempt to inhibit the response on occasional NoGo trials. Logan et al. (1984) conceptualised cancellation processes as a horse-race between a process for movement execution and another for movement cancellation. If the Go process wins, then the movement is executed; but if the stop process wins then the movement is successfully inhibited. The outcome depends heavily on when the stop signal is given. Success in cancelling a movement is more likely when the signal to stop is given early in the trial. In keeping with the horse-race analogy, action ("going") and inhibition of action ("stopping") are thought to be distinct processes with different neural pathways (Aron and Poldrack, 2006). Going is relatively slow, but selective for the specific action prepared. Stopping is relatively fast, and acts in an all-or-none, non-specific way (Coxon et al., 2007). The stopping process has proved difficult to study since stop and NoGo trials often do not produce overt behaviour. The main focus in behavioural studies has been the estimation of the internal latency of stopping (the stop-signal reaction time or SSRT).

Nevertheless, direct measures of the processes triggered by a stop signal are in short supply, because these processes produce no overt behaviour. The latency of stopping can only typically be inferred mathematically from models such as the horse-race model (Logan et al., 1984). Recently however, progress has been made in developing novel behavioural methods to measure the SSRT. For example, Morein-Zamir et al. (2006; 2007) developed a task where subjects tracked a visual target by manually pressing on a force sensor (the force-over time profiles provided a measure of the SSRT on every trial). However, this task involves subjects continuously adjusting an ongoing response and then having to stop *after* the action has been underway for some time.

Experiments 2 and 3 provided evidence that preparation can result in sensory suppression (see also Voss et al., 2006). The question now arises as to how long it takes the sensory system to recover when a prepared action is inhibited and stopped. In the current study, we propose to use the S-D (sensory-detection) task to reveal the internal latency and trajectory of stopping on trials where no movement or muscle activity is involved. The current experiment investigated the time-course of events triggered by the NoGo signal. On NoGo trials, when the prepared movement must be cancelled, we charted the dismantling of the now obsolete motor command by using weak cutaneous stimuli to reveal the time-course and trajectory of sensory detection at different intervals after the NoGo signal. A second aim was to map premovement sensory suppression onto an information processing stage. According to motor information processing theories (e.g. Crammond and Kalaska, 2000; Donders, 1969), the go process starts with the onset of the task stimulus and is thought to include the perceptual stage (processing of the Go or NoGo signal), the decisional stage (to go or not to go), and the motoric stage (preparation and execution of the appropriate movement; see Figure 5.1). If sensory suppression is elicited by decisional processes, then we would expect to see sensory suppression after a decision is made, regardless of whether the decision is to execute (i.e. Go trials) or to inhibit (i.e. NoGo trials) a movement.

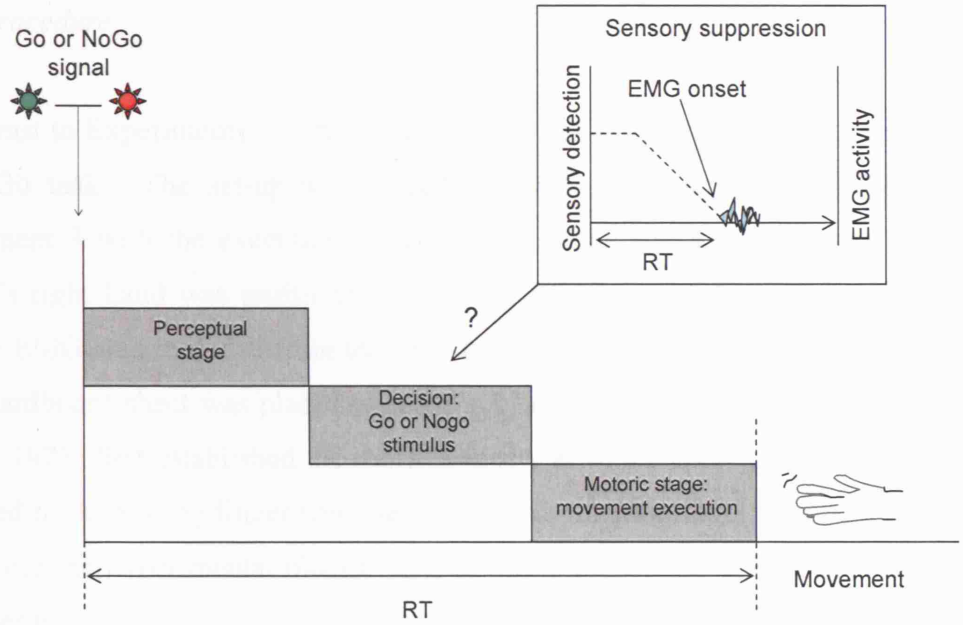

Figure 5.1. It is proposed that in the interval between the Go signal and the onset of movement (the reaction time) a series of stages takes place that includes the perceptual stage, the decisional stage and the motoric stage. However, it is not known which stage or stages cause sensory suppression.

In brief, we aim to i) use sensory suppression to measure the time-course of stopping on trials where no overt movement is involved and ii) investigate what stage, or stages, of processing in the preparation-execution chain is responsible for sensory suppression.

5.3 Materials and Methods

5.3.1 Subjects

Fourteen naïve subjects took part in the study. The data from 3 subjects were excluded because of unstable detection rates (post-test detection varied more than +/-15% from pre-experiment levels). Data from the remaining 11 subjects (7 female, 3 left-handed, mean age 23 years) were included in the final analysis.

5.3.2 Procedure

In contrast to Experiments 1-3, here there is no *prior instruction* task i.e. we only use the Go/NoGo task. The set-up and procedures were identical to the Go/NoGo task in Experiment 3 with the exception of the timing of the shock (see below). Briefly, the subject's right hand was positioned with the index finger resting on the pivoting plate. Surface EMG was recorded from the first dorsal interosseous (FDI) muscle as before. A black cardboard sheet was placed over the subject's right hand. The staircase procedure (Levitt, 1971) first established the shock intensity at which approximately 90% of stimuli delivered to the resting finger were detected. This intensity level then remained constant throughout the experimental blocks. The staircase procedure was repeated at the end of the experiment.

The start of each trial was marked by an acoustic signal. The green LED 1000 ms later signalled 'Go' for 6 subjects and the red light signalled 'NoGo', while for the remaining subjects this order was reversed. Subjects made speeded right index finger abductions in response to the 'Go' signal on movement trials, but withheld movement following the NoGo signal. After each trial, subjects reported verbally ('yes'/'no') whether they perceived a shock stimulus. No feedback was given. The experimenter then initiated the next trial after an intertrial interval of at least 1 second. After a practice block, subjects completed 6 experimental blocks of 54 trials. Each block consisted of 36 Go trials and 12 NoGo trials. A further 6 catch trials (no shock stimulus) were divided equally between Go and NoGo trials. The order of the trials was randomised. Shock stimuli were delivered either at the Go or NoGo signal, or after a 100 or 200 ms delay (see Figure 5.2).

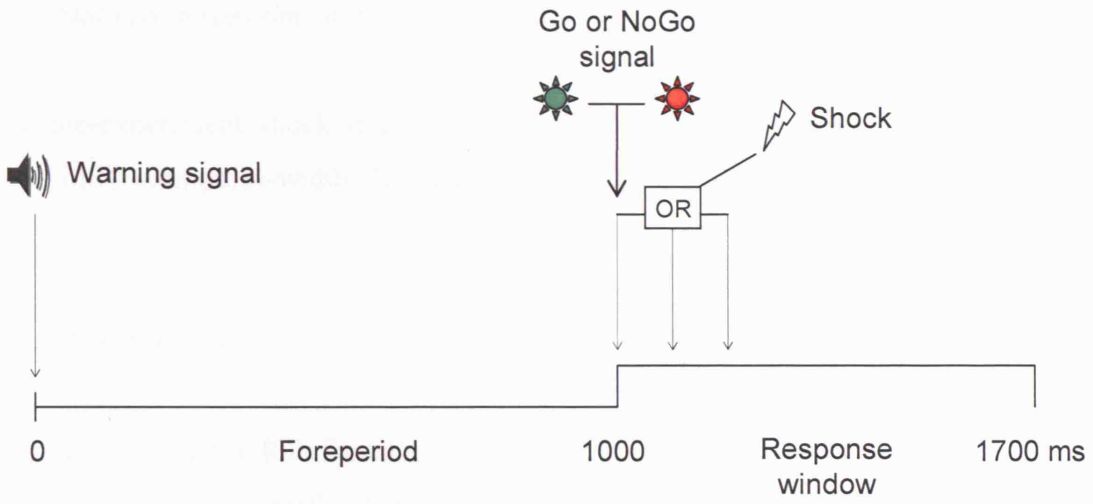

Figure 5.2. Experimental design. An acoustic warning signal marked the start of the trial. The Go or NoGo signal was presented after a delay of 1000 ms. Subjects waited for and responded to the Go signal, but withheld movement following the NoGo signal. The shock was delivered either simultaneously with the Go or NoGo signal or after a delay of 100 or 200 ms.

5.4 Results

Only 2.2% and 3.1% false positive detections occurred for Go and NoGo catch trials respectively. The number of false positives did not differ between trial types; $t(10)=0.559$; $p=0.588$ (see Table 5.1). The overall error of commission rate was 5.1%. Errors of omission accounted for 1.4% of all Go trials. Error trials were excluded from the analysis of sensory suppression effects.

Table 5.1. The number of false positive catch trials pooled across subjects for Go and NoGo trials.

Trial Type	
Go	4
NoGo	6

5.4.1 Stability across time of perceptual performance at rest

The pre-experiment shock intensity threshold did not differ from the post-experiment threshold (mean pulse-width=22.0 μs); $t(10)<0.001$; $p=1.000$ (two-tailed).

5.4.2 Reaction times

Prior to analysis, the RTs for each subject were trimmed as before, removing 4.6% of Go trials. The mean overall RT for Go trials was 291 ms (SD=75 ms). On 97.9% of movement trials the shock stimulus was delivered prior to the onset of EMG activity and the remaining 2.1% of trials were discarded.

5.4.3 Shock detection

In order to validate the behavioural paradigm used in this experiment, it is necessary to demonstrate that premotor sensory suppression is present. Therefore, the data were pooled from all subjects, trials were grouped into 50 ms bins relative to EMG onset and the graph plotted (Figure 5.3). The classic sensory suppression curve was revealed, replicating Experiments 1-3 and Williams et al. (1998). Weak cutaneous shocks were less likely to be detected just prior to onset of EMG activity and movement. A linear regression equation confirmed time-dependent changes in performance with a slope significantly different from zero; ($p<0.001$).

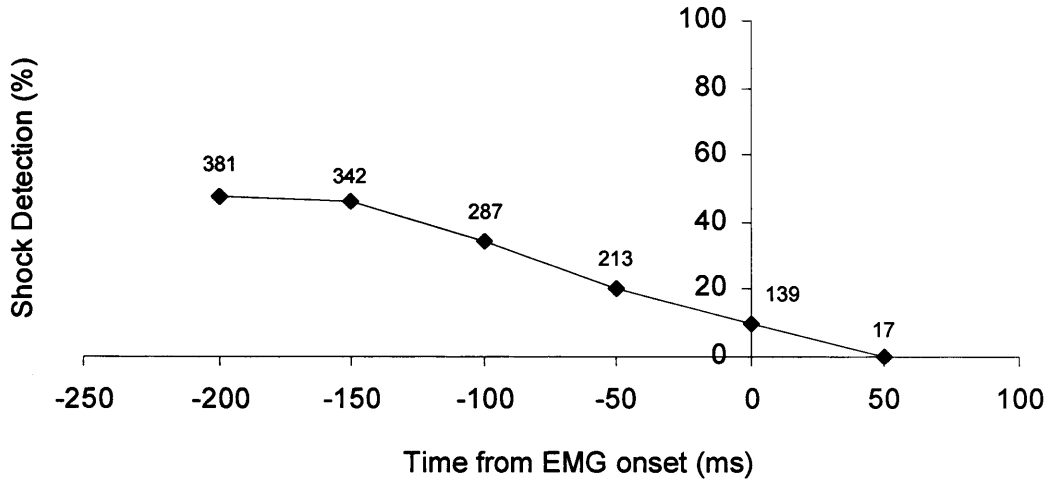

Figure 5.3. Effects of index finger movement on the detection of fixed intensity stimuli applied to the moving finger during Go trials. Detection performance over time is plotted relative to the onset of EMG (0 ms); a negative number indicates that the shock precedes EMG onset. The values next to the data points refer to the number of trials represented at that data point.

Figure 5.4 shows shock detection rates as a function of time. Note that the shocks were less likely to be detected closer to the onset of voluntary movement i.e. 200 ms after the Go signal as opposed to simultaneous with the Go signal (black bars). There was an opposite effect i.e. a recovery from sensory suppression with shocks most likely to be detected 200 ms after the NoGo signal as opposed to simultaneous with the NoGo signal (white bars).

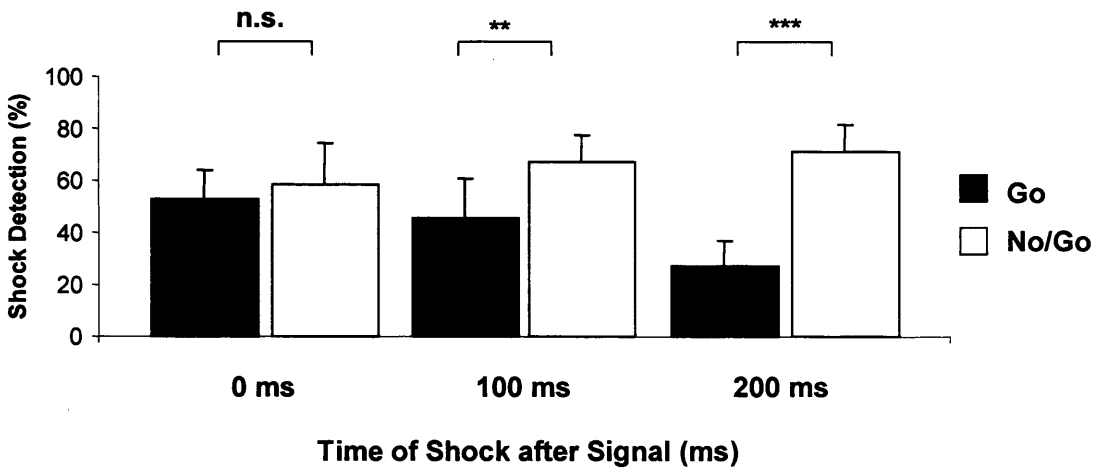

Figure 5.4. The mean percentage of stimuli perceived detected (+/- SD) for Go and NoGo trials. The shocks were delivered either simultaneously with the Go or NoGo signal or after a delay of 100 or 200 ms.

Shock detection rates were analysed with a repeated measures 2 x 3 ANOVA for the factors Go ('Go' vs. 'NoGo') and stimulus timing (0, 100 or 200 ms after the Go/NoGo signal). As expected, there was a significant main effect of movement $F(1,10)=20.997$; $p=0.001$. The main effect of stimulus timing was not significant $F(2,20)=2.796$; $p=0.102$, but the interaction between movement type and stimulus timing was significant $F(2,20)=35.325$; $p<0.0001$. This interaction was explored by holding each factor fixed in turn, and investigating the simple effect of the other factor. Two separate 3 x 1 ANOVAs performed on shock detection rates revealed significant effects of stimulus timing for the Go trials $F(2,20)=20.843$; $p<0.0001$ and interestingly, also for the NoGo trials $F(2,20)=5.450$; $p=0.015$ (values are two-tailed). Follow-up t tests were used to explore the effect for the NoGo trials. The difference in detection rates between 0 and 100 ms was significant $t(10)=2.591$; $p=0.027$. However, the difference between 100 ms and 200 ms did not reach significance $t(10)=0.848$; $p=0.416$ (values are two-tailed). Comparisons between conditions at each time interval revealed no difference between Go and NoGo trials at 0 ms, $t(10)=0.961$; $p=0.359$. However, comparisons for the later 100 and 200 ms intervals revealed significant differences $t(10)=3.376$; $p=0.007$ and $t(10)=7.514$; $p<0.0001$ respectively.

Models of action inhibition e.g. the horse-race model, assume that the stop process is faster than the go process (Logan, 1984; DeJong et al 1995; Coxon et al., 2007). An interesting question is therefore whether the development of sensory suppression on Go trials is faster than recovery from suppression on NoGo trials. Absolute change in detection rates was compared for Go and NoGo trials, for the intervals 0-100 ms, and 100-200 ms post-signal. Over the early 0-100 ms interval, the change in sensory suppression on Go trials was of similar size to the "dismantling" or recovery of suppression on NoGo trials $t(10)=0.452$; $p=0.661$. However, the absolute change in detection rates for the later interval, 100 to 200 ms, showed that sensory suppression on Go trials developed more quickly than recovery of sensation following NoGo signals $t(10)=2.272$; $p=0.046$.

5.5 Discussion

As in the previous experiments, weak cutaneous shocks were less likely to be detected just prior to a voluntary movement than when no movement occurred. This replicates sensory suppression effects previously reported (Experiments 1-3; Williams et al., 1998; Chapman and Beauchamp, 2006). Here, we studied the time-course of the recovery from sensory attenuation in the absence of overt movement. Interestingly, detection rates on NoGo trials improved monotonically as the interval between the NoGo signal and the shock was increased from 0 to 200 ms. We suggest that this recovery of sensation following a NoGo signal arises as follows. Up until the time of the NoGo signal, the movement is held in preparation and the sensory system is suppressed correspondingly (Voss et al., 2006). Once the NoGo signal is registered, the cortical motor pathway is gradually inhibited (stopping process), and the suppression of the sensory system is gradually released. We use the term 'dismantling the motor command' or 'dismantling' to refer to these processes together. The release from sensory suppression following the NoGo signal offers a new way to measure the stopping process. Our results suggest that stopping is not a single discrete event, but rather is a process that develops gradually and monotonically over time, taking approximately 200 ms to complete. The resulting view

of stopping is consistent with studies using other methods, such as the horse-race model (Logan et al., 1984). The horse-race model can only make inferences about the stopping process from those trials where subjects fail to stop and produce overt responses. Also, the horse-race model is concerned exclusively with the start and finish times of the stop process and makes no assumptions about the internal structure of stopping. Here, we have studied the stopping process by assessing how action-related suppression of sensation is dismantled over time as a result of a signal instructing cancellation of a prepared action.

As going and stopping are thought to involve independent processes according to the horse-race model, one might ask whether these processes proceed at different rates. Methods such as the horse-race model cannot measure these rates of progress directly. In contrast, we were able to test whether the development of sensory suppression following a Go signal occurred as rapidly as the dismantling of sensory suppression following a NoGo signal. For the first 100 ms, the attenuation after a Go signal and the dismantling of suppression following a NoGo signal proceed at similar rates. However, for the subsequent interval, 100 to 200 ms post-signal, the rate of movement-related sensory suppression on Go trials exceeds the rate of dismantling of suppression on NoGo trials. This rate difference could arise at a motor or a sensory stage. For example, a motor explanation based on horse-race models could hypothesise that the stop process operates more slowly than the go process. Alternatively, a sensory explanation could hypothesise that modulations of sensory cortex take longer to dissipate than to develop. Our own data do not allow us to distinguish between these two alternative hypotheses. Nevertheless, the stopping process may develop more slowly over time than the go process. Interestingly, this result contrasts with the assumption of psychological 'horse-race' models of stopping, which assume that the stop process is *faster* than the go process (Logan, 1984; DeJong et al., 1995; Coxon et al., 2007). Further investigation of this point would be a fruitful topic for future research.

A second aim of this Experiment was to try and map sensory suppression effects onto one of the central information processing stages (Crammond and Kalaska, 2000; Donders,

1969; Wolpert, 1997). We raised the question as to whether sensory suppression might map onto the decisional stage (see Figure 5.1). The decision here is between Go and NoGo. Specifically, we predicted that if the decisional process results in sensory suppression, then we would expect to observe sensory suppression effects once the decision is made, regardless of whether the decision is to execute or inhibit a movement. However, the results are clear. Sensory suppression only occurred when the decision resulted in overt movement (Figure 5.4; black bars), confirming that sensory suppression is related to the preparation and execution of movement. On the other hand, *recovery* from sensory suppression occurred when movement was successfully withheld (Figure 5.4; white bars). Sensory suppression arises downstream of the decisional stage.

In conclusion, the sensory correlates of stopping were measured revealing a recovery from sensory suppression. The recovery from sensory suppression can offer a new way of measuring the processes triggered by a NoGo signal. The recovery was significant 100 ms after the NoGo signal. There was evidence of further residual, non-significant recovery for the later 100 to 200 ms interval after the signal. Our results suggest that fully dismantling a prepared motor command takes at least 200 ms.

Experiment 4(b)

5.6 Abstract

A short follow-up experiment was carried out to examine the speed of onset of the dismantling (i.e. recovery from sensory suppression) process. The shock was delivered at 0, 25 and 50 ms after the NoGo signal. Detection rates improved as the interval increased from 0 up to 50 ms. The onset of the recovery from sensory suppression is fast, beginning in the first 25 ms after the NoGo signal. On Go trials, the shock was delivered 50 ms before subjects' mean reaction time in order to demonstrate sensory suppression.

5.7 Introduction

An advantage of the S-D task is that it can be used to measure the internal structure of stopping at any point during the stopping process. This is in contrast to the horse-race model which is only concerned with the start and stop times of the 'race'. Here, we adjust the timing of the shocks after the signal in order to determine the latency of the onset of dismantling. In Experiment 4(a), we mapped the recovery from sensory suppression from 0 to 200 ms (see Figure 5.4 and Figure 5.5(a). Specifically, shock detection rates were 58%, 68% and 71% at 0, 100 and 200 ms after the NoGo signal respectively. However, as sampling only took place at 0, 100 and 200 ms after the NoGo signal, any delay in the onset of dismantling lasting less than 100 ms would not be detected. Here, we have changed the resolution of the shock-timing to 0, 25 and 50 ms in order to "zoom in" on the beginning of the stopping process to determine whether the onset of dismantling is rapid [Figure 5.5(b)] or slow [Figure 5.5(c)].

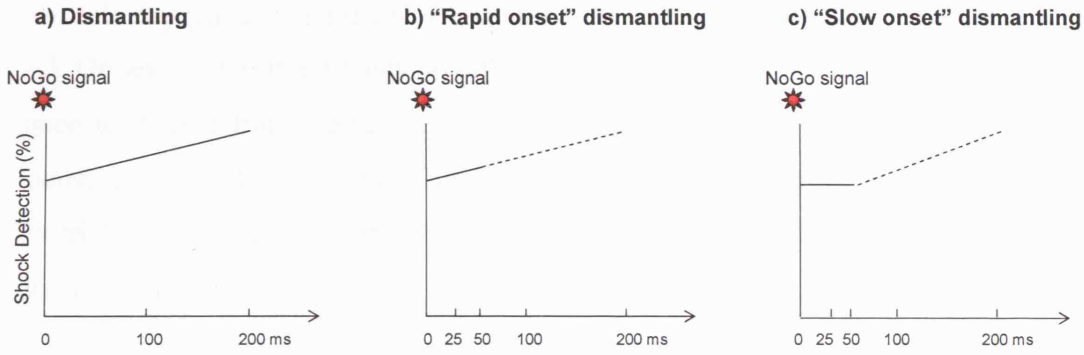

Figure 5.5. a) "Dismantling" as demonstrated in Experiment 4(a) (see also Figure 5.3) showing a steady increase in detection rates. b) First hypothesis for the current experiment showing "rapid onset" dismantling characterised by an upward slope in detection rates immediately after the NoGo signal and c) Second hypothesis showing "slow onset" dismantling where there is little or no recovery of shock detection rates (horizontal line) in the first 50 ms after the NoGo signal.

5.8 Materials and Methods

5.8.1 Subjects

Sixteen paid subjects took part with ethical committee approval. The data from 1 subject was excluded because their detection of cutaneous shocks at rest was unstable across the experiment (post-test detection varied more than +/-15% from pre-experiment levels). Data from the remaining 15 subjects (8 female, 12 right handed, mean age 27.8 (SD=9.5) years were included in the final analysis.

5.8.2 Procedure

After a practice block, subjects completed 6 experimental blocks of 44 trials. Each block consisted of 30 Go trials and 10 NoGo trials. A further 4 catch trials (no shock stimulus) were divided equally between Go and NoGo trials. The order of trials was randomised. Note in this experiment that the shock-timings for NoGo and Go trials are very different. Indeed, Go and NoGo trials focus on rather different questions. First, the NoGo trial was designed to demonstrate the nature of the onset of the release from sensory suppression by focusing on the first 50 ms of the dismantling process (Figure 5.6). Therefore, on NoGo trials, shock stimuli were delivered either at the NoGo signal, or after a 25 or 50 ms delay (Figure 5.6).

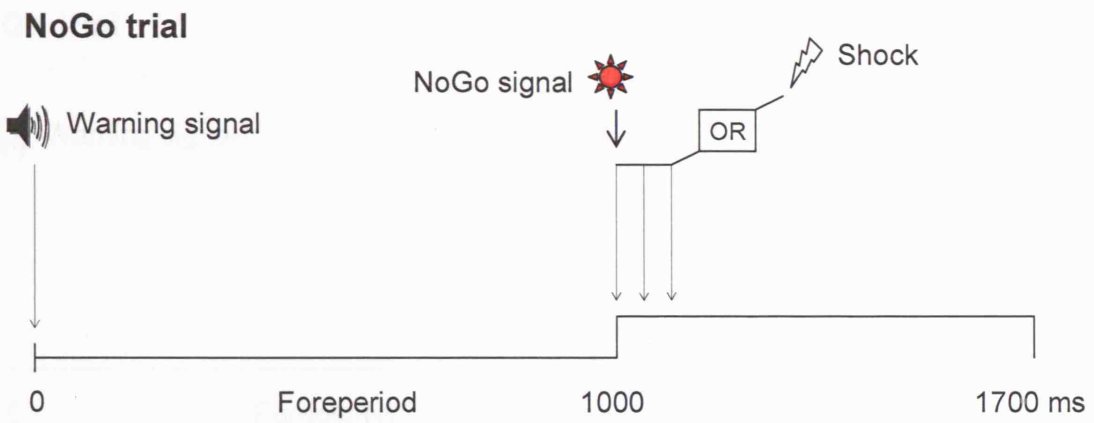

Figure 5.6. Experimental design for a NoGo trial. An acoustic warning signal marked the start of the trial. After a delay of 1000 ms, the NoGo signal was presented. Subjects withheld movement following the NoGo signal. The shock was delivered at 0, 25 or 50 ms after the NoGo signal.

Second, the Go trial was designed to demonstrate that premotor sensory suppression occurred in this paradigm. Therefore, on Go trials, shock stimuli were delivered 50 ms before each subject's mean RT calculated in the practice block at the start of the experiment (Figure 5.7). In contrast to Experiment 4(a), shock intervals were blocked with 2 blocks for each shock interval. The order of the blocks was interleaved for condition and the order of the trials was randomised.

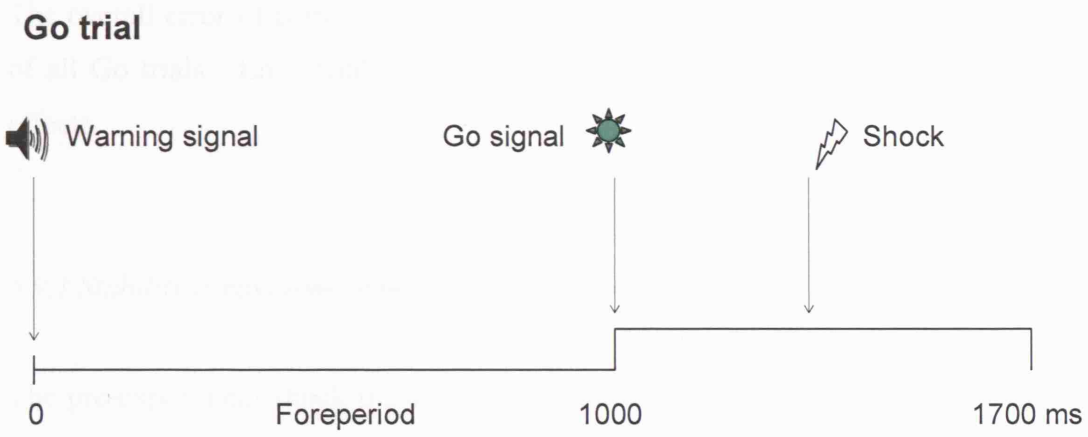

Figure 5.7. Experimental design for a Go trial. An acoustic warning signal marked the start of the trial. After a delay of 1000 ms, the Go signal was presented. Subjects waited for and responded to the Go signal. The shock was delivered at each subject's mean reaction time minus 50 ms. Note that the shock-timing is very different from that on NoGo trials (see Figure 5.6 above).

5.9 Results

Only 2.2% false positives and 0% false positive detections occurred for Go and NoGo catch trials respectively. The number of false positives did not differ between trial types; $t(14)=1.740$; $p=0.104$ (see Table 5.2).

Table 5.2. The number of false positive catch trials pooled across subjects during Go and NoGo trials.

Trial Type	
Go	4
NoGo	0

The overall error of commission rate was 4.2%. Errors of omission accounted for 1.6% of all Go trials. Error trials were not included in the analysis for sensory suppression effects.

5.9.1 Stability across time of perceptual performance at rest

The pre-experiment shock intensity threshold (mean pulse-width=23.5 μs) did not differ significantly from the post-experiment threshold (mean pulse-width=21.8 μs); $t(14)=1.163$; $p=0.264$.

5.9.2 Reaction times

Prior to analysis, the RTs for each subject were trimmed as before, removing 3.6% of Go trials. The mean overall RT for Go trials was 338 ms (SD=57 ms). On 64.6% of movement trials the shock stimulus was delivered prior to the onset of EMG activity. The remaining 35.4% of trials, on which the shock occurred after EMG onset, were not considered for analysis of premotor sensory suppression effects.

5.9.3 Shock detection

In order to validate the behavioural paradigm, the data were pooled from all subjects, trials were grouped into 50 ms bins relative to EMG onset and the graph plotted (Figure 5.8) demonstrating time-dependent changes in the detection of stimuli applied to the moving digit and confirming premovement sensory suppression. A linear regression equation confirmed time-dependent changes in performance with a slope significantly different from zero; ($p=0.015$).

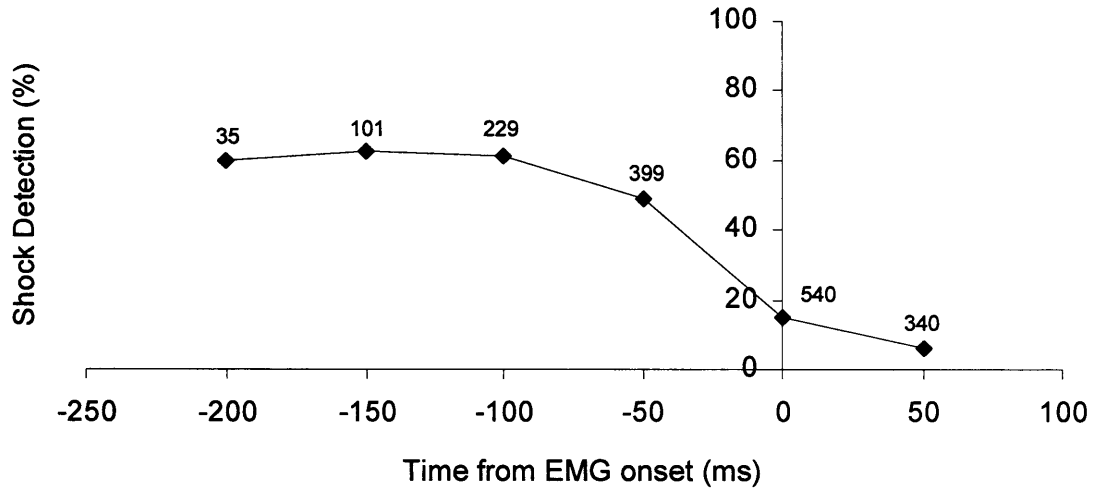

Figure 5.8. Effects of index finger movement on the detection of fixed intensity stimuli applied to the moving finger during the Go trials. Detection performance over time is plotted relative to the onset of EMG (0 ms); a negative number indicates that the shock precedes EMG onset. The values next to the data points refer to the number of trials represented at that data point.

Shock detection rates for the NoGo trials are presented in Figure 5.9. Note that shock detection performance improves from 0 to 50 ms. Shock detection rates were subjected to a 1 x 3 ANOVA for the three shock delivery times (0, 25 or 50 ms after the NoGo signal). There was a main effect of stimulus timing $F(2,28)=6.208$; $p=0.008$. Follow-up t tests were used to explore this effect. The overall difference in detection rates between 0 and 50 ms was significant $t(14)=3.916$; $p=0.001$ (one-tailed). The difference in detection rates between 0 and 25 ms was also significant $t(14)=2.046$; $p=0.030$ (one-tailed). However, the difference between 25 ms and 50 ms did not reach significance $t(14)=1.126$; $p=0.149$ (one-tailed).

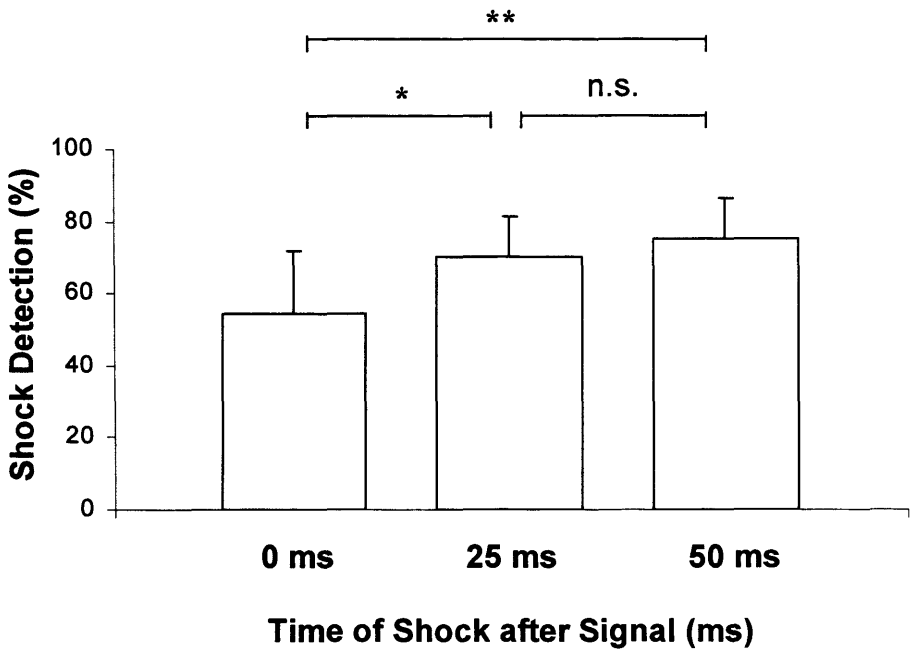

Figure 5.9. The mean percentage of stimuli detected (+/- SD) for NoGo trials. The shocks were delivered either simultaneously with the NoGo signal or after a delay of 25 or 50 ms.

5.10 Discussion

The results of Experiment 4(b) are clear. The onset of the dismantling process is fast, in line with the "rapid onset" hypothesis (Figure 5.5.b). The sensory system adjusts immediately once a prepared movement has been cancelled. Furthermore, subjects clearly showed sensory suppression on Go trials (see Figure 5.8). Taken together, the results from Experiment 4(a) and (b) suggest that dismantling a prepared motor command takes approximately 200 ms. The onset of the dismantling process is rapid, occurring within the first 25 ms after the NoGo signal.

Chapter 6: Sensory suppression in the "stop-signal paradigm"

6.1 Abstract

In the last chapter, we have shown that during a Go/NoGo task going and stopping have distinct sensory signatures. Here, we combined the sensory-detection (S-D) task with the stop-signal paradigm in order to investigate what happens to sensation when motor processes for going and stopping are both simultaneously active as in the "horse-race" of motor control. Subjects were required to abduct their right index finger as quickly as possible after the presentation of the Go signal. In addition, subjects had to stop the response if an occasional stop-signal was presented. After the Go signal, the stop-signal timing was adjusted so that subjects could stop successfully on approximately 50% of stop trials. On trials where no stop signal occurred, normal premovement sensory suppression effects were observed. However, on stop-respond trials, where subjects were unable to inhibit movement successfully and responded despite the stop signal, there was an unexpected upward inflection in shock detection performance. The inflection in the sensory suppression curve was suggestive of the sensory signature of stopping, even though subjects did not stop on those trials. Sensory performance, as measured by rates of shock detection, clearly depended on whether the stop processes were active or not and not only on the Go process that drove movement initiation. When Go and Stop processes are placed in direct competition against each other, there is a brief time window when the sensory system is in stop mode while the motor system is in Go mode. The observed pattern of sequential sensory activation suggests that stopping and going are not mutually exclusive and independent (cf., Logan et al., 1984).

6.2 Introduction

When performing everyday activities, people can withhold, interrupt, or rapidly modify planned or ongoing actions when these actions are suddenly rendered inappropriate by unanticipated changes or events. This ability to dynamically adjust one's actions to the changing demands of the environment is of theoretical interest as it can provide an important source of information about the nature of executive processes that dynamically regulate the operations of the human information-processing system. The preparation and execution of movement appear to depend on an intricate and, at present, poorly understood interplay between excitatory and inhibitory mechanisms by which the cognitive executive system and the motor system interact.

In the laboratory, the stop-signal paradigm is the best established method for studying these cognitive-motor interactions. In the stop-signal paradigm, the subject is usually engaged in a primary reaction time task requiring the subject to press a key with the index

finger as quickly as possible after the presentation of the reaction stimulus. In addition to this primary task, the subject has to stop the response if an occasional stop-signal is presented. The chances of successful stopping depend on the interval between go-signal and stop-signal presentation, referred to as the *stop-signal delay* (SSD). Stopping is easy when the stop-signal is presented shortly after the primary task signal but becomes increasingly difficult, or virtually impossible, when the stop-signal delay is increased and approaches the moment of response execution. A trial is labeled *stop-inhibit* if the subject successfully inhibits the response that would have been produced otherwise. A trial is labeled as *stop-respond* if the subject is unable to inhibit the response. Typically, as SSD increases, subjects' ability to inhibit the response decreases, so the probability of stop-respond trials increases (Logan and Cowan 1984; van den Wildenberg et al., 2006).

The stop-signal task has been used extensively to study executive control and flexibility in behaviour. Numerous experimental manipulations of the stop-signal task have yielded very similar results, demonstrating the generality of the task as an empirical model of self-control and the uniform nature of the stop process. Stop signals have ranged from visual (e.g., Lappin and Eriksen, 1966) to auditory (e.g. Logan, 1981; Logan et al., 1984) to tactile (e.g. Akerfelt et al., 2005). Responses have included key presses (e.g. Logan et al., 1984), typing responses (e.g. Logan, 1982), speech output (e.g. Ladefoged et al., 1973), arm movements (e.g. McGarry et al., 2003), hand squeezes (e.g. De Jong et al., 1995), hand movements (e.g. Boucher et al., 2007b) and eye-movements (e.g. Hanes and Carpenter, 1999).

A unified and widely accepted account of stopping behaviour is provided by the horse-race model. This model assumes that performance is the outcome of a race between a go process responsible for initiating the movement and a stop process responsible for inhibiting the movement (Logan and Cowan, 1984). These processes are assumed to be independent. According to the race model, behaviour is governed by the finish times of the go and stop processes on a given trial. A response is initiated if the go process reaches threshold first; a response is inhibited if the stop process reaches threshold first. The Go signal triggers the Go process and the later stop-signal triggers the stop process.

Go and stop processes may proceed at different rates. Hence a stop process beginning after a go process may nevertheless reach threshold first thereby successfully canceling behaviour. The race model can account for the distribution of stop-respond RTs by relating the proportion of stop-respond trials to the distribution of RT on trials with no stop-signal in the following manner. When no stop-signal is presented, the full RT distribution is produced. When a stop-signal occurs, only a fraction of the RT distribution is produced because only the fastest RTs can escape inhibition.

Beyond articulating an account of the functional mechanism responsible for performance, the race model provides a mathematical means for estimating the time needed to inhibit a response, referred to as the *stop-signal reaction time* (SSRT; Logan and Cowan, 1984). This is important because the response to the stop-signal is not directly observable. If the stop process is quicker than the go process, there is no overt response for which latency can be measured. However, if the stop process is slower than the go process, a response occurs, and we know therefore that the stop RT must have been longer than the latency of the response - however, we do not know how much longer. The race model provides a method for estimating SSRT from the no-stop-signal RT distribution (De Jong et al., 1990; Logan and Cowan, 1984). The horse-race model assumes that the SSRT is constant. Though this assumption is unlikely to be strictly true, computer simulations (Band et al., 2003; De Jong et al., 1990) have shown that the assumption of constant SSRT does not bias the estimation substantially. The method is illustrated in Figure 6.1.

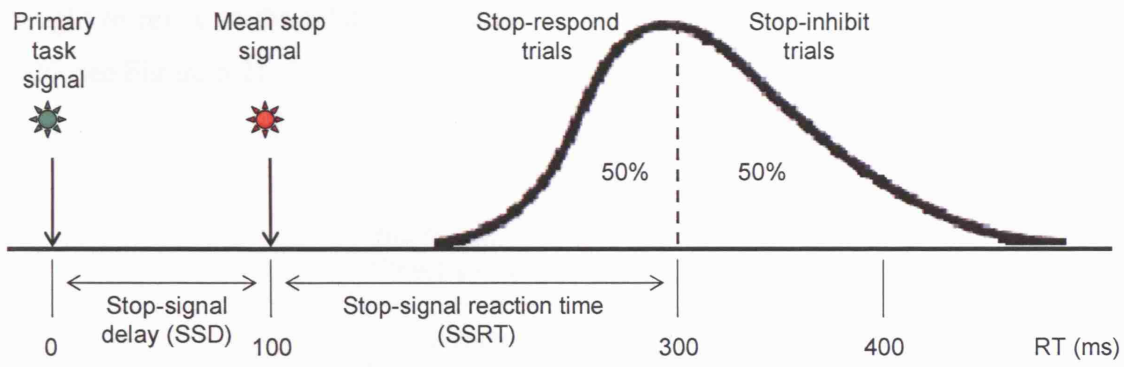

Figure 6.1. Estimation of stop-signal reaction time (SSRT) according to the horse-race model (Logan and Cowan, 1984). Typical values (ms) are shown. The curve depicts the distribution of RTs on no-stop-signal trials (trials without a stop signal) representing the finishing times of the response processes. Assuming independence of go and stop processes, the finishing time of the stop process bisects the choice RT distribution (dashed vertical line). Given that the movement could be withheld in 50% of all stop trials, stop-signal RT (200 msec) is calculated by subtracting the mean stop-signal delay (100 msec) from the mean no-stop-signal RT (300 ms).

The finishing time of the stop process divides the no-stop-signal RT distribution into two parts, one in which the no-stop-signal RT is less than the SSRT and one in which the no-stop-signal RT is greater than the SSRT. The area under the no-stop-signal RT distribution corresponding to the left part (Figure 6.1) equals the proportion of stop-respond trials, and the area under the no-stop-signal RT distribution corresponding to the right part equals the proportion of stop-inhibit trials. Thus, the SSRT is estimated by finding the point that divides the no-stop-signal RT distribution into these two parts and subtracting the SSD (Logan et al., 1984).

Go and stop processes have been linked to different neural pathways (Goldman-Rakiç, 1987). More recently, a functional MRI (magnetic resonance imaging) study demonstrated that going significantly activated frontal, striatal, pallidal and motor cortical regions (Aron and Poldrack, 2006). This pattern of activation is consistent with the "direct" pathway (Nambu et al., 2002). Stopping significantly activated right inferior frontal cortex (IFC) and the subthalamic nucleus (STN; Aron and Poldrack, 2006) via the "hyperdirect" pathway (Nambu et al., 2002). The speed of Go and Stop processes is

thought to relate to the relative activation of these neural pathways (Aron and Poldrack, 2006; see Figure 6.2).

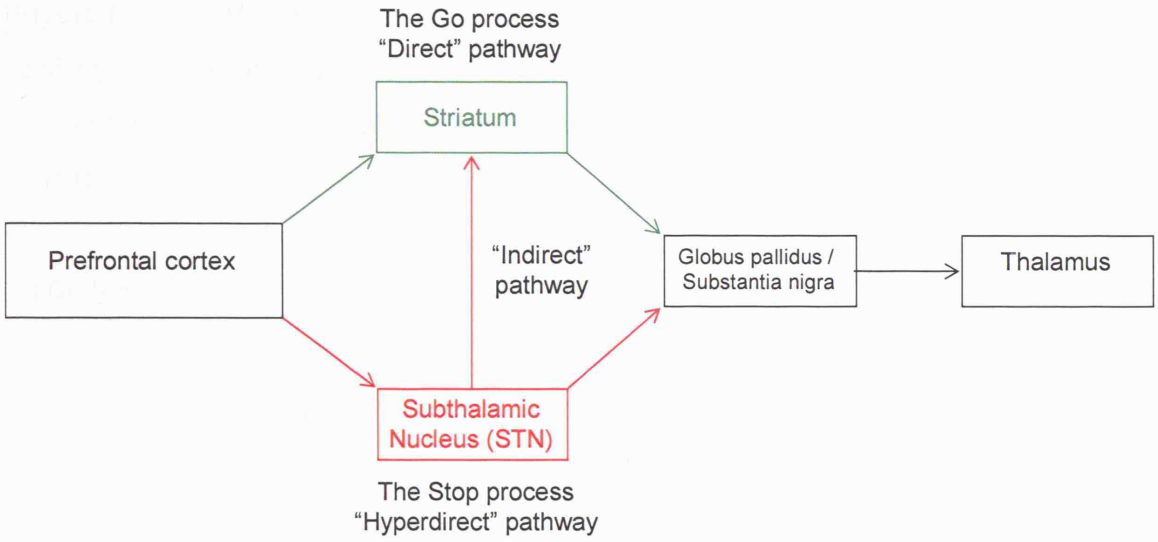

Figure 6.2. A basal-ganglia model. An influential model proposes the go and stop processes compete via "direct" and "hyperdirect" pathways through the basal-ganglia. The balance of stopping and going processes is thought to select action or action inhibition (Figure after Nambu et al., 2002).

It has been suggested that the STN, which is part of the basal ganglia, may suppress the "direct" fronto-striatal pathway that is activated by response initiation. Basal ganglia models suggest that STN activation could occur via the "hyperdirect" fronto-subthalamic pathway or via the "indirect" fronto-striatal-pallidal-subthalamic pathway (Alexander and Crutcher, 1990; Mink, 1996; Nambu et al., 2002). Recent work has specifically implicated the STN in stop-signal response inhibition. STN stimulation improves SSRT in patients with Parkinson's Disease (van den Wildenberg et al., 2006) and midbrain lesions that damaged STN slowed SSRT in rats (Eagle and Robbins, 2003).

The two paradigms used to investigate motor preparation, execution and inhibition processes, namely the Go/NoGo task (e.g. Hoshiyama et al., 1996) and the stop-signal paradigm (e.g. Logan et al., 1984), differ in terms of the amount of competition generated

between the go and stop processes. On Go and NoGo trials there is no explicit competition between movement plans. Thus, on a single trial, a Go signal triggers overt movement *or* a NoGo signal results in the cancellation of the prepared movement, presumably activating the "direct" go *or* the "hyperdirect" stop pathway respectively [Figure 6.3(a)]. However, this is not the case on a stop trial where the movement plans cued by the Go *and* the subsequent presentation of a stop-signal, are placed in *direct competition* presumably resulting in the coactivation of the go and stop pathways [Figure 6.3(b)].

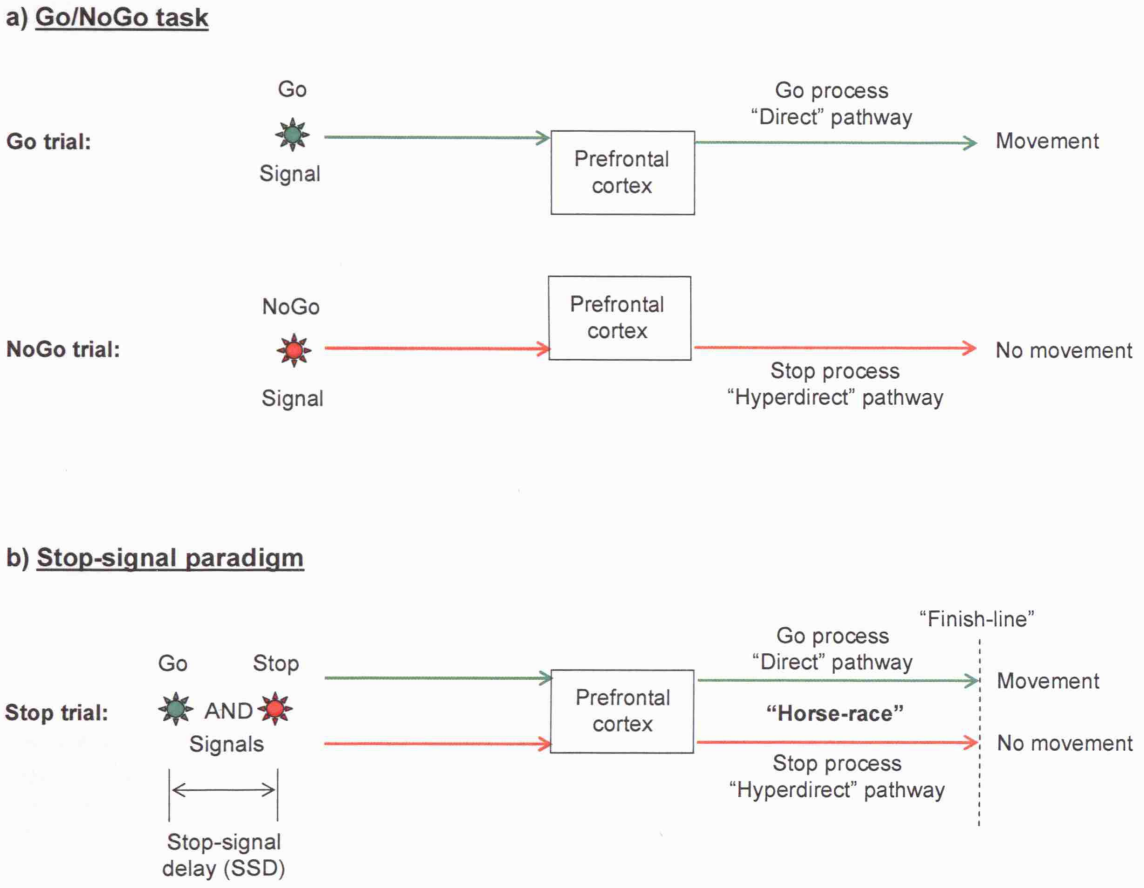

Figure 6.3. Schematic of Go, NoGo and stop trials in the Go/NoGo and stop-signal paradigms. The prefrontal cortex (Goldman-Rakiç, 1987) shunts the commands generated by the visual signals into the appropriate response. a) During the Go/NoGo task, the response is either to go *or* not to go resulting in activation of the go ("direct") or the stop ("hyperdirect") pathway respectively. b) The stop-signal paradigm. On a stop trial, the stop-signal is presented at a variable time delay (called the "stop-signal delay" or SSD) after the Go signal. Thus, the activation of the go process *competes* directly against the *coactivated* stop process in a "horse-race" towards a winner-takes-all threshold "finish-line" that determines whether a movement is executed or not.

The level of competition between the go and stop processes is critically maximised by adjusting the delay between the presentation of the Go signal and the stop-signal (i.e. the SSD) during the course of the experiment as performance varies, so that a successful change of plan occurs on approximately half of stop trials.

It has previously been shown that going and stopping have distinct sensory signatures [Experiments 4(a) and (b); Walsh and Haggard, 2007]. Specifically, in a Go/NoGo task, sensory suppression increases just prior to movement (Figure 6.4, green line; Williams et al., 1998) but recovers when we cancel a prepared movement (Figure 6.4, red line; Walsh and Haggard, 2007).

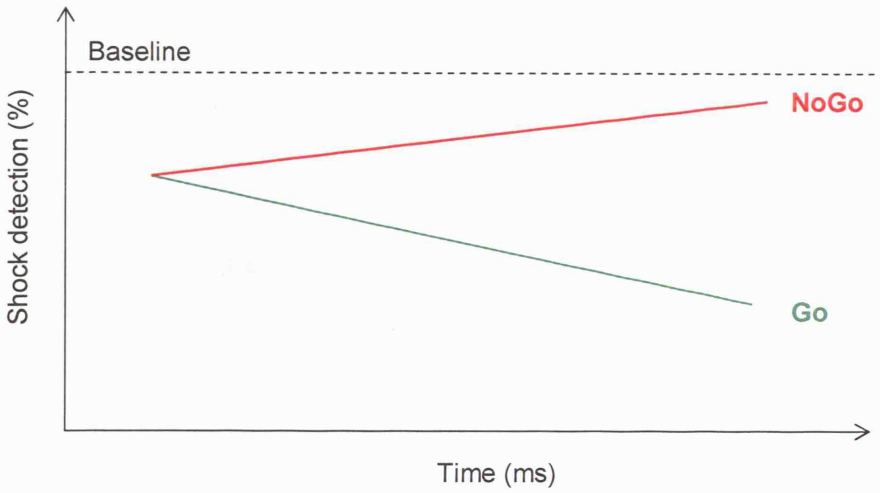

Figure 6.4. Stopping and going have distinct sensory signatures [redrawn schematically from Experiment 4(a)]. Going is associated with a decrease in shock detection rates prior to movement i.e. premovement sensory suppression (green line). In contrast, stopping is associated with an increase in shock detection rates or a recovery from sensory suppression (red line) towards baseline detection rates.

Here, we ask what will happen to sensation in the stop-signal paradigm (Logan et al., 1984) at the moment when independent go and stop processes are placed in maximum competition against each other. Stop-respond trials, where subjects are unable to inhibit movement successfully, are of central interest. Sensory suppression on such trials could, in principle, be driven by the go process, the stop process, or by a mixture of both. If the

signal driving the sensory suppression effect is dominated by the Go process, then we predict the sensory signature of going prior to movement on stop-respond trials (Figure 6.5 (a1); "100% Go"). In contrast, if the sensory suppression effect is dominated by the stop process, this would result in the sensory signature of stopping (Figure 6.5 (a3); "100% Stop"). Alternatively, go and stop processes may interact sequentially as indicated by sensory suppression effects that show the combined serial influence of the Go and Stop processes [Figure 6.5 (b4 and b5)].

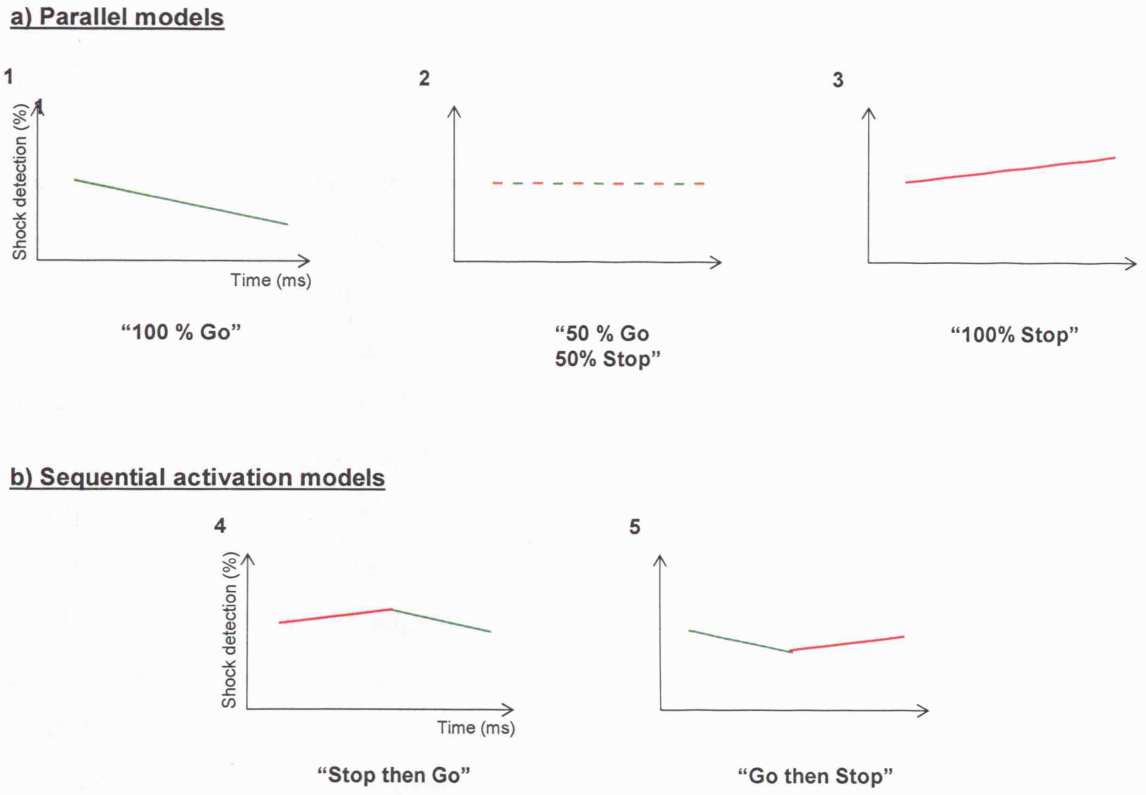

Figure 6.5. Schematic of possible interactions between go and stop processes during a stop trial.
a) Parallel models:
1. The go process is dominant, resulting in the sensory signature of going ("100% Go").
2. The go and stop processes average out ("50% Go, 50% Stop").
3. The stop process is dominant, resulting in the sensory signature of stopping ("100% Stop").
b) Sequential activation models:
4. First, the stop process is dominant, followed by the go process ("Stop then Go").
5. First, the go process is dominant, followed by the stop process ("Go then Stop").

6.3 Materials and Methods

6.3.1 Subjects

Eleven paid subjects took part. Data from all 11 subjects were included in the final analysis; Four were female, 9 were right-handed and the mean age was 26.4 (SD = 7.1) years.

6.3.2 Procedure

A brief acoustic tone marked the onset of the trial. Subjects were presented with one of three trial types at random (Figure 6.6). After 1000 ms, on no-stop-signal trials, the green Go signal was presented for 700 ms. On NoGo trials, a red signal was presented for 700 ms. On stop trials, the go signal was presented for an interval (either 60, 80, 100, or 120 ms) and was then replaced by the stop-signal for the remainder of the response window. Typically in the stop-signal paradigm, the stop-signal delay (SSD) algorithm is adjusted on a trial-by-trial basis. Here, the SSD was adjusted after every four stop-trials, as follows. The first block started with an initial stop-signal delay of 100 ms. At the end of each block of 18 trials, the algorithm worked by subtracting 20 ms from the previous value of the stop-signal latency if more than two responses were correctly withheld, and added 20 ms to it when less than two full responses were produced. If exactly half of responses were correctly withheld and half were executed, then the stop-signal delay value was kept constant. In this way, probabilities of approximately 50% responses and 50% correct inhibitions can theoretically be obtained. In keeping with the behavioural definition of reaction time and response completion, stop trials were classified as stop-respond trials if full movement ensued (i.e. if the potentiometer output was equal to 20°). If the potentiometer output was greater than 0° and less than 20°, the response was classified as a "partial-respond" trial. If there was no movement i.e. potentiometer output = 0°, and no EMG activity, the trial was classified as a stop-inhibit trial.

During no-stop-signal trials, shock stimuli were delivered at each subject's mean RT calculated during the practice block, minus 50 ms or minus 120 ms [Figure 6.6(a)]. Shocks were delivered at these two intervals in order to obtain an RT distribution in the critical interval for sensory suppression just prior to movement onset (Williams et al., 1998). The timing of the shock stimulus on NoGo trials was the same as on no-stop-signal trials [Figure 6.6(b)]. On stop trials, shock stimuli were delivered at each subject's mean RT minus 100 ms or minus 170 ms [Figure 6.6(c)]. Shock timing was based on the results of a pilot study (not reported here) where stop-respond trials yielded RTs that were on average 50 ms faster than no-stop-signal trials. The aim was to try and match shock delivery times relative to EMG onset for the no-stop-signal and the stop-respond trials in order to be able to compare rates of sensory suppression for both trial types, despite differences in RT.

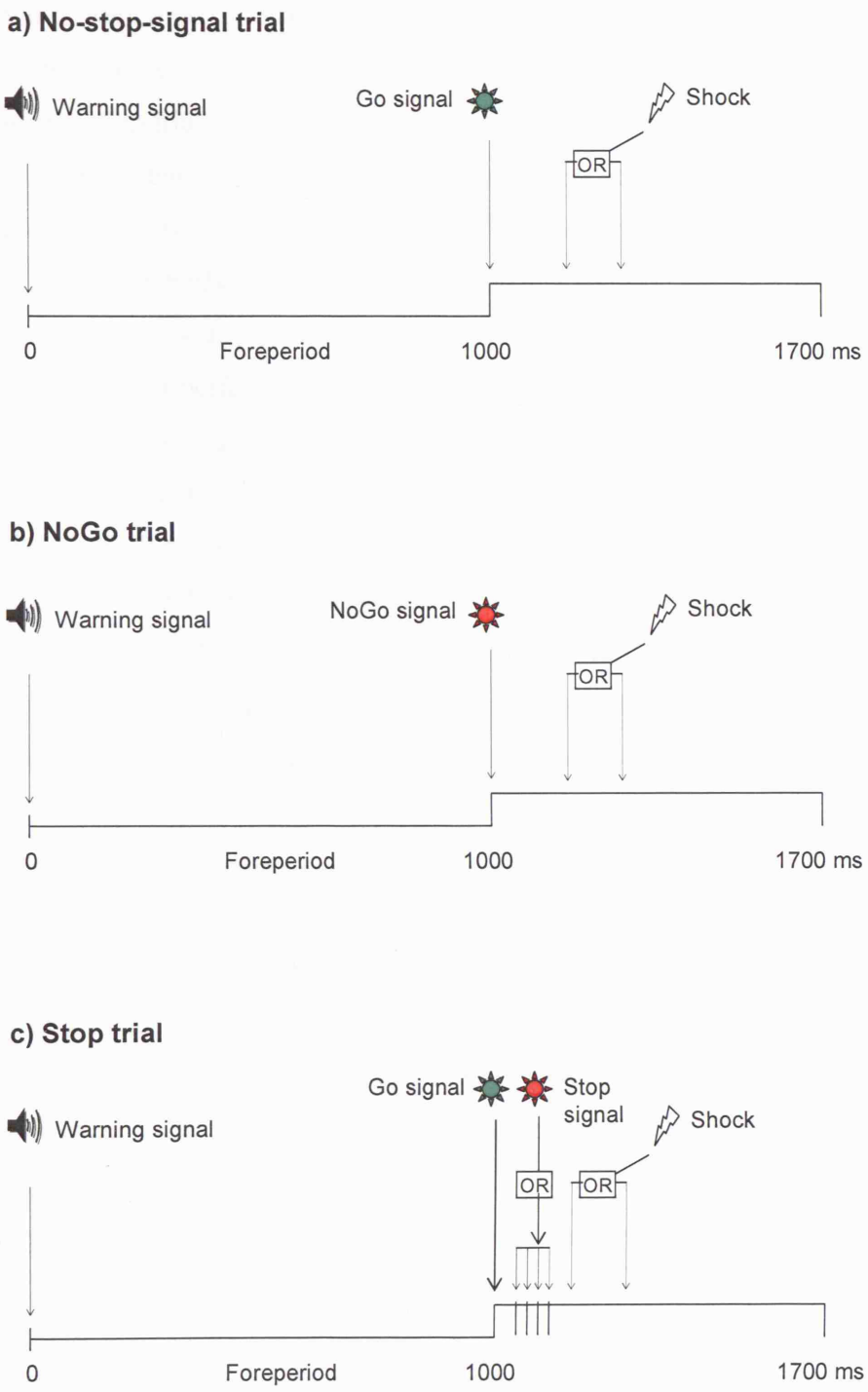

Figure 6.6. Trial types. After an acoustic warning signal the (a) Go or (b) NoGo signal was presented after 1000 ms. Subjects waited for and responded to the Go signal, but withheld movement following the NoGo signal. On no-stop-signal trials, the shock was delivered at each subject's mean reaction time minus 50 or 120 ms. Shock timing on NoGo trials was identical to no-stop-signal trials. (c) On stop trials, the Go signal was followed by the stop-signal after a delay of 60, 80, 100 or 120 ms, adjusted in response to a staircase procedure. On stop trials, the shock was delivered at each subject's mean reaction time minus 100 or 170 ms.

As in previous experiments, subjects made speeded right index finger abductions in response to the Go signal on no-stop-signal trials, but were instructed not to move on NoGo trials. On stop trials, subjects were asked to try and withhold their response once a stop-signal occurred, but were told that this would not be possible on all of the stop trials. Stopping and going were given equal emphasis. After each trial, subjects reported verbally ('yes'/'no') whether they perceived a shock stimulus. No feedback was given. Trials were self-paced with a minimum intertrial interval of 1 second before the start of the next trial. Subjects performed a practice block of 44 trials (no-stop-signal and NoGo trials only) during which the subject was trained to respond quickly and consistently and during which their mean RT was established. This was followed by 19 blocks. Each block consisted of 18 trials including 10 no-stop-signal trials; 2 NoGo trials and 4 stop trials. A further 2 catch trials, comprising one movement and one stop catch trial, contained no shock stimulus. The order of the trials was randomised. The first block was considered practice and not analysed. Subjects were given the opportunity to take short breaks between blocks throughout the experiment.

After completing the 19 experimental blocks, subjects completed a further two "Go-control" blocks. These blocks acted as a control showing movement performance and shock detection in the absence of NoGo and stop trials. These served to show the extent of sensory suppression associated with the Go process. There were 18 Go-control trials in each block (including 2 non-shock catch trials). These trials were identical to no-stop-signal trials [Figure 6.6(a)]. However, it was made clear to subjects that these two blocks would not contain stop-signal nor NoGo trials. They were asked to wait for the green Go signal and then to respond as quickly and consistently as possible.

6.4 Results

On catch trials 4.3% false positive detections were recorded. The number of false positives did not differ between no-stop signal and stop-signal trial types; t(10)=1.336; p=0.211 (see Table 6.1).

Table 6.1. The number of false positive catch trials pooled across subjects during no-stop-signal and stop trials.

Trial Type	
No-stop-signal	6
Stop	11

Errors of omission (i.e. movement trials without movement during the response window) occurred on 1.4% of movement trials. These trials were excluded when measuring the effects of sensory suppression. Errors of commission (i.e. when a NoGo trial was accompanied by movement and / or EMG activity) were 10.1%. The rate of errors of commission was unexpectedly high. However, 48% of these errors involved partial movement or electromyographic activity with no movement, confirming that subjects were attempting to inhibit responses.

6.4.1 Stability across time of perceptual performance at rest

The pre- and post-experiment staircases showed similar shock intensity thresholds (mean pulse-widths=22.5 and 22.2 μs: t(10)=0.260; p=0.800.

6.4.2 Reaction Times

On stop-signal trials, 55% of stop trials were successfully inhibited (i.e. stop-inhibit trials) involving no EMG activity and no potentiometer reading. The remaining 45% (stop-respond trials) involved either full (25% of stop-signal trials) or partial (20% of stop-signal trials) movement. Reaction times for each subject were trimmed to +/- 2 SD (excluding 4.6% of movement trials). The mean RT on no-stop-signal trials was calculated for each subject. An estimate of the SSRT was obtained by subtracting out the stop-signal delay for each subject from their mean reaction time (Logan, 1984). Because the stop-signal delay was a blocked factor, this analysis was performed separately for every block, and the estimated values were then averaged across blocks. The mean stop-signal delay was = 96 ms. Given the mean RT of 299 ms, this gives a value of 203 ms for the SSRT.

Consistent with the horse-race model, the mean RT on stop-respond trials (266 ms; SD=64 ms) was shorter than the mean RT on no-stop-signal trials (299 ms; SD=57 ms); $t(10)=4.354$; $p=0.001$. When subjects respond very fast, they are unable to inhibit the response to the stop signal. Therefore, trials classified as stop-respond trials tend to be fast. This seems to be a very robust finding in stop-signal tasks (e.g. van Boxtel et al., 2001; De Jong et al., 1995; Jennings et al., 1997; Logan, 1984). The finding agrees with the intuitive notion that stop-respond trials are those trials that were fast enough to escape inhibition, corresponding to the leftmost part of the no-stop-signal RT distribution (Figure 6.7).

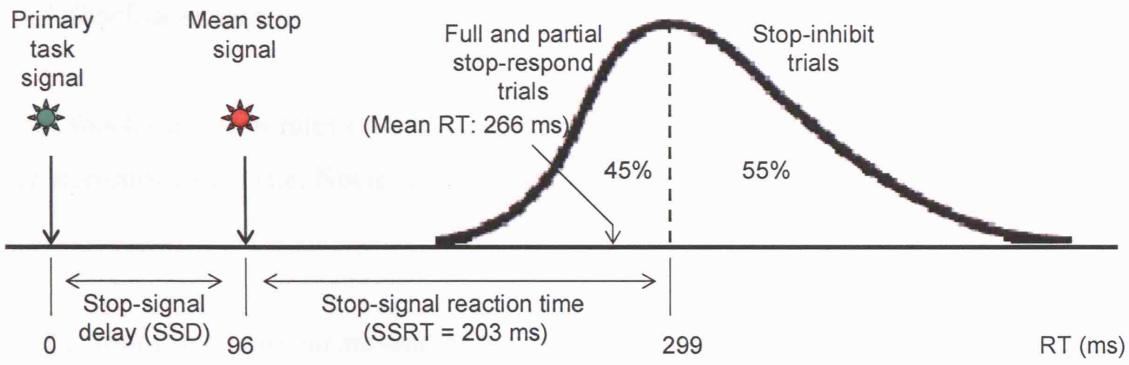

Figure 6.7. Results showing the estimation of stop-signal reaction time (SSRT) according to the horse-race model (Logan, 1994; Logan and Cowan, 1984). The curve depicts the distribution of RTs on no-stop-signal trials (trials without a stop signal) representing the finishing times of the response processes. Assuming independence of go and stop processes, the finishing time of the stop process is equal to the mean no-stop-signal RT. Stop-signal RT (203 msec) is calculated by subtracting the mean stop-signal delay (96 msec) from the mean no-stop-signal RT (299 ms).

On 87.8% of movement trials the shock stimulus was delivered prior to the onset of EMG activity. The remaining 12.2% of trials were discarded from the sensory suppression analysis.

Go-control trials were incorporated into the design as a control showing reaction time performance in the absence of NoGo and stop trials. The present data indicate that processing of the stop-signal interfered with primary-task performance, thus violating the stop-independence assumption of the race model. The mean reaction time on Go-control trials was 223 ms (SD=40 ms). This RT is 76 ms faster than the mean RT on no-stop-signal trials (299 ms). This suggests that subjects strategically waited when performing the stop-signal paradigm. Subjects were instructed not to slow their response in order to enhance their chances of being able to inhibit the response when required. Despite our efforts to prevent this strategy, the Go-control trial results show that some such slowing occurred, demonstrating that subjects adopted a tonic inhibitory motor set during performance of the stop-signal paradigm. This is a common finding in these studies (e.g. Logan et al., 1986).

6.4.3 Shock detection

First, shock detection rates on movement (i.e. no-stop-signal and stop-respond) trials and then non-movement (i.e. NoGo and stop-inhibit) trials will be analysed.

6.4.3.1 Shock detection on movement (i.e. no-stop-signal and stop-respond) trials

Stop-respond and partial-respond trials were classified together when analysing premotor sensory suppression as both types of trials involved overt movement and EMG activity. Furthermore, shock detection rates for stop-respond and partial-respond trials did not differ statistically; $t(10)=0.874$; $p=0.403$.

The mean shock to EMG-onset intervals for no-stop-signal and stop-respond trials were 104 ms (SD=62 ms) and 120 ms (SD=71 ms) respectively. However, this difference just reached significance $t(10)=2.296$; $p=0.045$ indicating that our attempts to time the shock in an equivalent way across trial types was not entirely successful. Subjects were slightly slower on stop-respond trials than the 50 ms difference observed in the pilot study. Although the two types of trials were not perfectly matched for mean shock-EMG onset asynchrony as a whole the variation in RTs means that there is a substantial overlap between the shock-EMG onset asynchrony distributions. Selecting trials from each type with matching shock-EMG onset asynchrony i.e. response-locking the data, therefore allows a fairer comparison of sensory suppression. Previously, response-locking of the data was not necessary as there was no difference in shock-EMG onset asynchrony. Here, shock-EMG onset asynchrony did vary. Therefore it was necessary to recalculate the shock-EMG onset asynchrony on every trial, in order to produce a strictly response-locked data set.

Before re-binning the data, we first plotted the graph for data pooled from all subjects to study the time-course of any variation in performance during movement. Trials were grouped into 50 ms bins relative to EMG onset and the graph plotted (Figure 6.8) for no-

stop-signal and stop-respond trials. For no-stop-signal trials, detection of shock stimuli diminished with increasing proximity to EMG in the usual manner (Experiments 1-4; Williams et al., 1998). Interestingly, on stop-respond trials, there was an unexpected upward inflection of shock detection rates starting 150 ms before EMG onset (Figure 6.8). This increase in shock detection rates is reminiscent of the dismantling process demonstrated in Experiment 5.

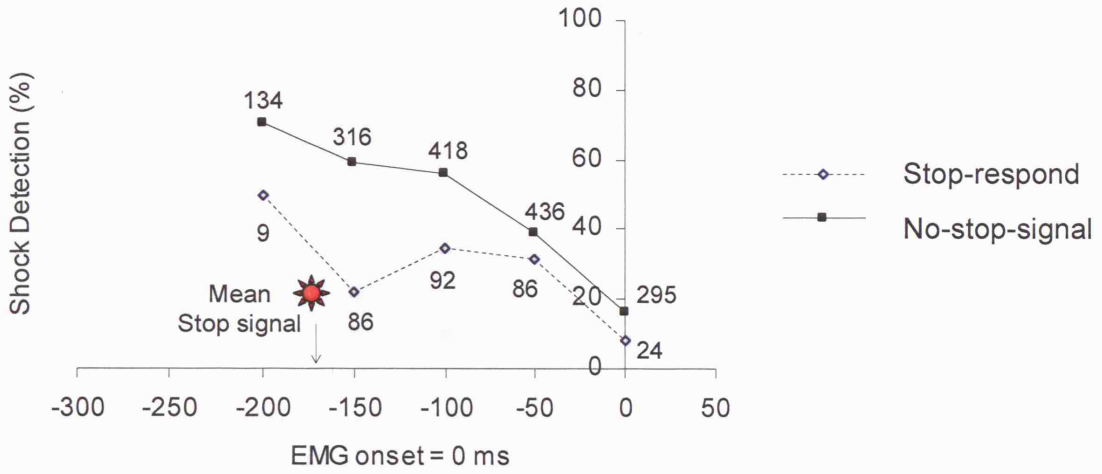

Figure 6.8. Effects of finger abduction on the detection of fixed intensity stimuli applied to the moving finger (50 ms bins) for no-stop-signal trials and stop-respond trials. Detection data are plotted over time relative to onset of EMG (0 ms). The mean RTs for the no-stop-signal and the stop-respond trials were 299 ms and 266 ms respectively. The values next to each data point refer to the number of trials the data point is based on. Data are pooled across all subjects.

In order to plot the graph for response-locked data, we then sorted the data for each subject into 50 ms time-bins relative to EMG onset in order to perform a repeated-measures analysis. We calculated the mean shock detection rate for each time-bin for each subject. In this way we could lock shock detection rates for each time-bin to the trial types to EMG onset thereby enabling statistical comparison between matched trials. This trial-matching method is not always perfect. While there are sufficient no-stop-signal trials in each time-bin to represent each subject, this was not always the case for the stop-respond trials. Thus, while eleven subjects were represented in the 150 ms, 100 ms and 50 ms bins, only eight subjects were represented in the 0 ms bin. For the remaining 3

subjects, values extrapolated from the difference in shock detection rates for the 100 and 50 ms time-bins were inserted in place of the missing values. In the time-bin 200 ms before and 50 ms after EMG onset there were insufficient trials to represent more than 3 subjects per bin and therefore these bins were disregarded from the statistical analysis. Based on the mean values, a new curve was constructed (Figure 6.9) allowing shock detection rates for no-stop-signal and stop-respond trials over the range -150 ms to 0 ms relative to EMG onset, to be compared statistically.

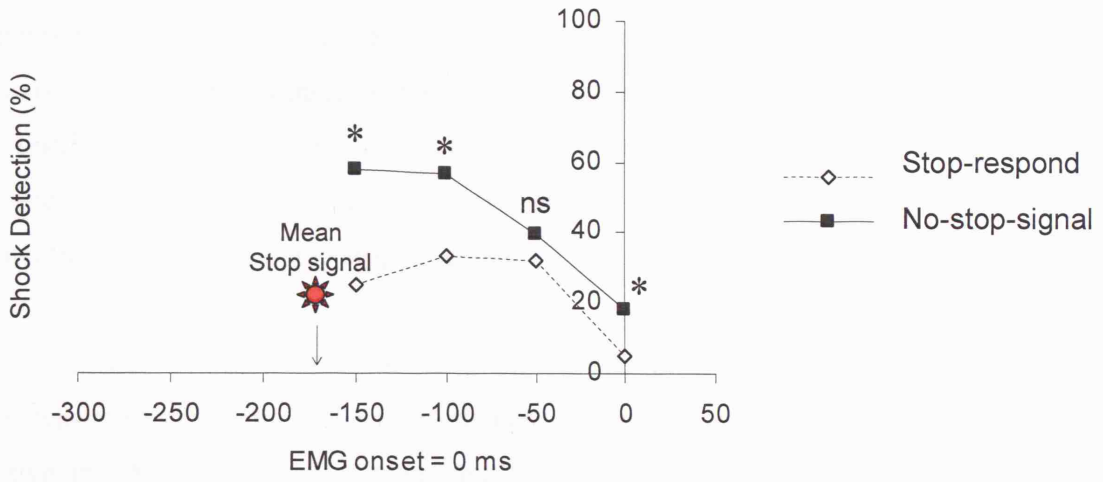

Figure 6.9. Mean shock detection rates for individual subjects for stop-respond versus no-stop-signal trials. The mean RTs for the no-stop-signal and the stop-respond trials were 299 ms and 266 ms respectively.

Figure 6.9 shows shock detection rates for the no-stop-signal and stop-respond trials for individual means. The no-stop-signal trials show the characteristic premotor sensory suppression decrease in shock detection rates prior to EMG onset. However, for the stop-respond trials the upward inflection in detection rates that starts 20 ms after the presentation of the mean stop-signal and lasts for 100 ms (time-bin 150 ms to 50 ms before EMG onset) was still present. Therefore, the inflection is not just an artefact of pooling the data. This subset of data points was subjected to a 2 x 4 repeated measures ANOVA for the factors trial type (no-stop-signal vs. stop-respond) and time-bin (150,

100, 50 or 0 ms before EMG onset). There was a significant effect of trial $F(1,10)=17.517$; $p=0.002$ and a main effect of time-bin $F(3,30)=15.118$; $p<0.0001$. Surprisingly, given the horse-race assumption of independence for go and stop processes, the interaction for trial type by time-bin was also significant $F(3,30)=3.621$; $p=0.034$. Follow-up t tests were used to explore the interaction. Shock detection rates were significantly higher on no-stop-signal trials relative to stop-respond trials for the 150 ms $t(10)=4.308$; $p=0.002$, 100 ms $t(10)=3.603$; $p=0.005$ and at 0 ms bins $t(10)=3.670$; $p=0.004$. However, there was no significant difference in shock detection rates between trial types at 50 ms before EMG onset $t(10)=0.929$; $p=0.375$; (Figure 6.9). According to the race model, the go and stop processes should not interact. The go process which initiates the movement and the stop process which inhibits the movement should not directly influence each other. The go process should drive sensory suppression on trials that result in movement irrespective of whether the stop process is active or not. It should be noted that the interaction in our data is between *sensory* variables that are assumed to reflect the underlying go and stop processes.

The difference between the slopes for the curves for no-stop-signal and stop-respond trials was explored using linear trend analysis. When the four time-bins (-150 ms to 0 ms relative to EMG onset) were examined, the difference between slopes was significant $F(1,10)=11.167$; $p=0.007$. This difference depended critically on the upward inflection in the earliest time-bin (-150 ms before EMG onset) of the stop-respond condition, since there was no significant difference when this bin was not included in the trend analysis $F(1,10)=1.504$; $p=0.248$. The shape of the curve for stop-respond trials was also explored using the curve estimation routine in SPSS to test whether a quadratic model provided a significantly improved fit over a linear model (see Table 6.2). In each case a one-tailed probability is given: for the no-stop-signal condition, progressive sensory suppression predicted a negative slope, while for the stop-respond condition, an initial release of sensory suppression following stop signals predicted a negative quadratic term (peak- rather than valley-shaped).

Table 6.2. A comparison of no-stop-signal and stop-respond trials using linear and quadratic models based on their relative goodness of fit. Note the large difference in the R-Square statistic (R^2) between the linear and quadratic models for stop-respond trials.

Trial Type	Model	R^2	p value (one-tailed)
Stop-respond	Linear	0.367	0.197
	Quadratic	0.971	0.085
No-stop-signal	Linear	0.890	0.029
	Quadratic	0.994	0.038

As can be seen from Table 6.2, there was a weak trend (p=0.085) towards significance for the quadratic model for stop-respond trials. More interestingly, the R-Square statistic (R^2) for the stop-respond trials is very low for the linear model (0.367), but very high for the quadratic model (0.971). Thus, adding the quadratic term captures the true shape of the curve in this condition, due to the initial upward deflection. For the no-stop-signal condition, the R-Square statistics for the two models are much closer.

6.4.3.2 Shock detection on Go-control trials

Go-control trials acted as a control showing movement performance and shock detection in the absence of NoGo and stop trials and served to show the extent of sensory suppression associated with the Go process. In order to validate the behavioural paradigm, the data were pooled from all subjects, trials were grouped into 50 ms bins relative to EMG onset and the graph plotted (Figure 6.10) demonstrating time-dependent changes in the detection of stimuli applied to the moving digit and confirming premovement sensory suppression. Note that the classic downward premovement sensory suppression slope is clearly present. A linear regression equation confirmed time-dependent changes in performance with a slope significantly different from zero; (p=0.006).

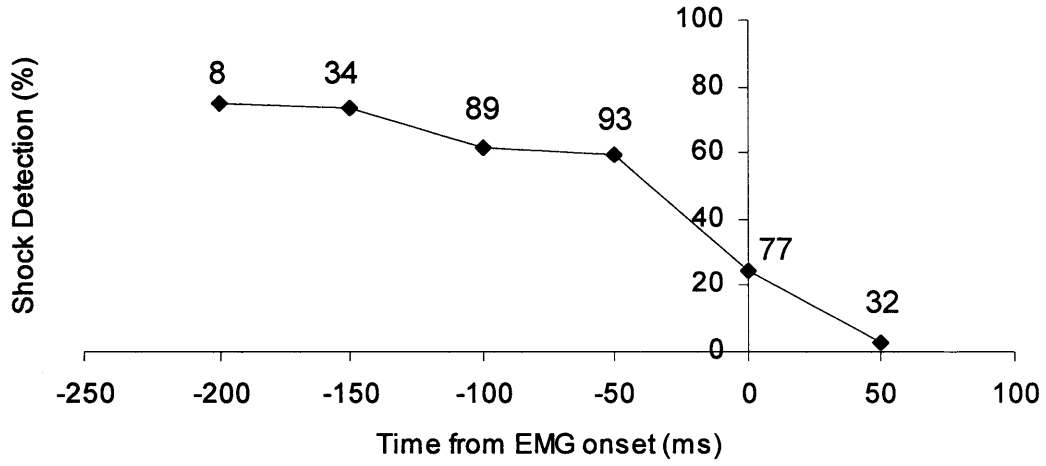

Figure 6.10. Effects of index finger movement on the detection of fixed intensity stimuli applied to the moving finger during the Go-control trials. Detection performance over time is plotted relative to the onset of EMG (0 ms); a negative number indicates that the shock precedes EMG onset. The values next to the data points refer to the number of trials represented at that data point.

6.4.3.3 Shock detection on non-movement (i.e. NoGo and stop-inhibit) trials

Shock detection performance on NoGo and stop-inhibit trials was investigated. The shock was randomly delivered at one of two intervals ("early" and "late") after the signal (see Figure 6.6). The mean shock detection rate on stop-inhibit trials was 61.5% (61% and 62% when the shock was delivered "early" and "late" respectively; see Figure 6.11). On NoGo trials, the mean shock detection rate was 72.5% (72% and 73% when the shock was delivered "early" and "late" respectively). The mean time of shock delivery on stop-inhibit trials was 147 ms ("early" mean delivery time = 112 ms; "late" mean delivery time = 182 ms) and 192 ms on NoGo trials ("early" mean delivery time = 157 ms; "late" mean delivery time = 227 ms). Shock detection rates were analysed with a repeated measures 2 x 2 ANOVA for the factors trial (NoGo and stop-inhibit) and stimulus timing ("early" vs. "late"). There was a main effect of trial type; $F(1,10)=7.774$; $p=0.019$. Shock detection performance was better on NoGo trials relative to stop-inhibit trials though on NoGo trials the shock was delivered on average 45 ms later. There was no main effect of

stimulus timing F(1,10)=0.133; p=0.723 and the interaction between trial and stimulus timing was also not significant F(1,10)=0.004; p=0.951.

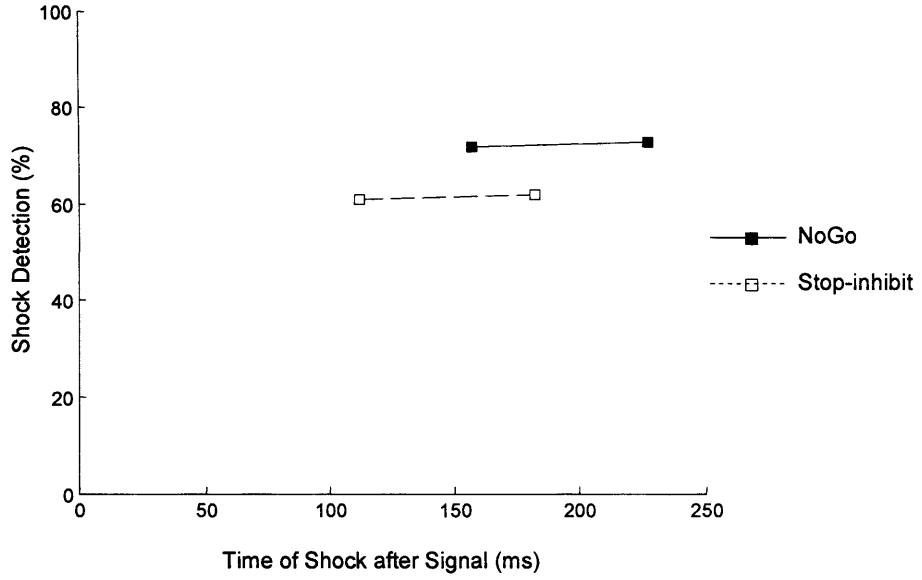

Figure 6.11. The mean percentage of stimuli detected for NoGo and stop-inhibit trials. The shocks were delivered randomly at one of two intervals ("early" or "late") after the NoGo or stop signal (see text for details).

6.4.3.4 "EMG-only" trials

In the current study, on a small subset of stop-inhibit trials (23 trials, pooled from 7 subjects), the agonist FDI muscle was activated (as measured by EMG) but no overt movement was recorded. These are labelled "EMG-only" trials here, and we report them as they are of theoretical interest (e.g. McGarry and Franks, 1997; De Jong et al., 1990). The combined use of potentiometer and EMG / FDI allowed us to detect these trials. Muscle activation must occur for an overt response to be executed. However, muscle activation can occur without movement if response processing can be interrupted after muscle activation has started but before the response has been completed. Response is made up of two processes; an early controlled process that can be inhibited, and a subsequent ballistic process that cannot be inhibited once it is started (Logan and Cowan, 1984). The point at which processing becomes ballistic represents the last possible site of

inhibition and is therefore of considerable theoretical interest as it identifies the point where the race ends (Osman et al., 1986; Osman et al., 1990). The presence of these trials suggests that subjects are able to control stopping up to a point downstream of activation of the agonist muscle and before movement. The mean shock detection rate on these trials was 56% with a mean shock delivery time of 117 ms (SD=60 ms) after the stop signal.

6.5 Discussion

Previously, we demonstrated during a Go/NoGo task that going and stopping have different sensory signatures (Chapter 5, Walsh and Haggard, 2007). Here, we extend this finding to the stop-signal paradigm where go and stop processes are placed in direct competition against each other. The stop-signal was adjusted so that subjects could stop successfully on approximately 50% of stop trials. Of special interest were sensory suppression effects during stop-respond trials. We compared response-locked sensory detection rates for stop-respond trials and corresponding no-stop-signal trials. On no-stop-signal trials, normal premovement sensory suppression effects were observed. However, on stop-respond trials there was an unexpected upward inflection in shock detection performance from 150 to 50 ms before EMG onset that is suggestive of the release from sensory suppression previously identified (Walsh and Haggard, 2007). Sensory performance, as measured by rates of shock detection, clearly depended on whether the stop process was active (stop-respond trials) or not (no-stop-signal trials) and did not depend only on the Go process that drove movement initiation. In summary, we observed a hybrid signature with the sensory pattern of stopping followed by the sensory pattern of going. Our results suggest that stop and go processes interact during countermanding behaviour, in a way consistent with the "Stop then Go" sequential activation hypothesis [see Figure 6.5(b.4)].

There are a number of alternative explanations for the upward inflection on stop-respond trials which can be discounted. Firstly, the inflection in sensory suppression could be a

reaction time effect on sensory performance. It is known that sensory performance can vary with movement kinematics (Angel and Malenka, 1982). However, this possibility is ruled out as the inflection of the sensory suppression curve should then also be present in the no-stop-signal trials which were also response-locked. This was not the case. A second possibility is that the inflection might have resulted from arousal triggered by the Go signal. This possibility can also be discarded as no-stop-signal trials which also contained a Go signal did not show the effect. Yet a third possibility is that the inflection resulted from arousal triggered by the *change* from the Go to the stop signal. This possibility is dismissed in the next experiment (see Chapter 7) when the order of the Go and stop signals is reversed. Instead, the inflection appears to be a specific consequence of the processes that would stop an ongoing response. Indeed, the upward trend of the curve (see Figure 6.9; -150 ms to -50 ms) recalls the dismantling of sensory suppression following a NoGo signal observed in Chapter 5. Once the stop signal is registered, the stopping process gradually inhibits the cortical motor pathway and gradually releases the suppression of the sensory system. An inspection of Figure 6.9 shows that shock detection rates on stop-respond trials are lower than on no-stop-signal trials. This may occur because on no-stop-signal trials the "direct" (go) pathway is highly activated while the "hyperdirect" (stop) pathway is deactivated. However, on stop-respond trials both the go and stop pathways are *coactivated.* Lower detection rates on stop-respond trials relative to no-stop-signal trials could result from the blocking of basal-ganglia thalamocortical output resulting from the coactivation of the go and stop pathways (Aron and Poldrack, 2006).

Measures of the stop-signal reaction time (SSRT) have been used extensively as indices of self-control across a variety of domains including human development (e.g., Bedard et al., 2002; aging (e.g., Kramer et al., 1994), and individual differences (e.g., Logan et al., 1997; Miyake et al., 2000). Clinical psychologists have used the SSRT to assess deficits resulting from diseases of the brain such as Parkinson's disease (Gauggel et al., 2004), and schizophrenia (Badcock et al., 2002). SSRT measures have been useful in understanding attention-deficit/ hyperactivity disorder (ADHD; see Nigg, 2001, for review). The SSRT is useful in clinical applications because it is a single datum that

measures the inhibitory ability of a subject. It can be correlated with other measures and used to distinguish the abilities of one group from those of another. A large body of research rests on the validity of the SSRT as a measure of inhibitory processing, which in turn, relies on the validity of the race model. In turn the race model makes the critical assumption that the stop and go processes proceed independently up until a finishing line where the winner takes all.

6.5.1 Interaction between go and stop processes

There is increasing neurophysiological evidence that the neural processes of excitation and inhibition that produce behaviour do, in fact, interact. For example during eye movement tasks, saccade initiation time is influenced by the balance of inhibition between fixation and movement neurons (Trappenberg et al., 2001). Observations like these are not consistent with the notion that behaviour appears to be the outcome of a race between processes with independent finishing times. In the horse-race model, the critical assumption is that the activation of one process (e.g. the stop process) has no effect on the second process (e.g. the go process; Logan and Cowan, 1984; Hanes and Carpenter, 1999).

Very recently, an *interactive* computational horse-race model has been proposed by Logan and colleagues (Boucher et al., 2007a) to try and explain this paradox. In the *interactive* horse-race architecture, the stop and go processes are seen as mutually inhibitory, so a stop-respond trial occurs if the go process reaches threshold, and a stop-inhibit trial occurs if the stop process prevents the go process from reaching threshold. According to the interactive horse-race model (Boucher et al., 2007a) the duration of the SSRT is occupied by the following events. First, during stimulus encoding, the stop process does not influence the go process, initially satisfying the independence premise of the original race model. Thus, it is proposed that the first and longest part of the SSRT duration is occupied by an independent race between the go and stop processes. Second, once activated the stop process potently and rapidly inhibits the go process; it is proposed

that this powerful inhibition process takes approximately 25 ms (Boucher at al., 2007a). Finally, a very short ballistic interval (approximately 10 ms) precedes the initiation of the movement (see Results Section 6.4.3; Logan and Cowan, 1984). According to the interactive horse-race model, the SSRT is the sum of these three intervals (Figure 6.12).

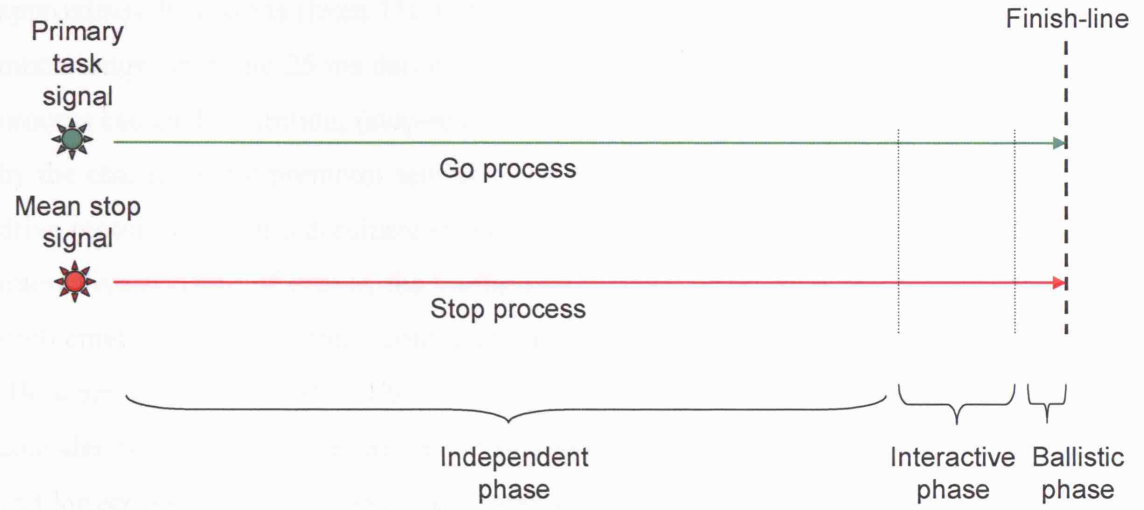

Figure 6.12. The interactive horse-race model after Boucher et al. (2007a). According to the model, the horse-race consists of three phases. First, in the independent phase, the go and stop processes compete against each other independently. This is the longest part of the race. Second, during the interactive phase, the very potent stop process interacts with the go process; this phase is proposed to last approximately 25 ms. The third and final ballistic phase is very brief (approximately 10 ms). In this phase the Go process can no longer be stopped.

While the horse-race model is concerned exclusively with the start and finish times of the go and stop processes, it makes no assumptions about the processes by which the RTs are generated beyond assuming they are stochastically independent (Logan, 1994). An advantage of the sensory-detection (S-D) task is that it can measure the *internal structure* of stopping (Walsh and Haggard, 2007) and is therefore a new and useful tool with which to investigate what happens *during* the race. Therefore, it can be used to evaluate the interactive horse-race model. The interactive model proposes that the initial part of the SSRT duration is occupied by an independent race between the go and stop processes.

According to the model, the stop process does not activate until the final "furlong" of the race and has an approximate duration of 25 ms (Boucher at al., 2007a). Crucially, our data suggest that the stopping process is activated much earlier than the model proposes and can act within 20 ms of the presentation of the stop-signal (see Figure 6.9), immediately influencing sensory processing by dismantling sensory suppression. Furthermore, the duration of the inhibition observed in the current study lasted for approximately 100 ms (from 150 to 50 ms before EMG onset; see Figure 6.9), a duration much longer than the 25 ms described by Boucher et al. (2007a). On trials where the go process escaped inhibition, (stop-respond trials), the final 50 ms of the race was occupied by the characteristic premotor sensory suppression curve as the go process "kicked in" to drive motor output and dominate sensation. Thus, while our results are consistent with an interactive account of events, the timing and duration of the stop process observed here is problematic for the current computational model described by Logan and colleagues (Boucher et al., 2007a). The timing in the Boucher model is based on theoretical considerations only. Here, we derive timing estimates from data. These show an earlier and longer interaction between the go and stop processes than the Boucher model, at least regarding the effects of the go and stop processes on sensation.

6.5.2 Independent interpretation of the data

It should be noted that the upward inflection in sensory processing observed on stop-respond trials (Figure 6.9) can also be explained in terms of an independent horse-race and a two stage process. In the following speculative, alternative interpretation of our data, one process controls behaviour while the other process controls sensation. In this race there are two finish-lines, a motor finish-line where a motor decision is taken and a later sensory finish-line where a sensory decision is taken. The race starts once the primary task (Go) signal is presented, thereby releasing the go process. The stop-signal is presented at the end of the stop delay, on average 96 ms after the Go signal, initiating the stop process. Both processes race against each other. The go process reaches the motor finish-line first, triggering a motor command. However, the stop process is faster and

more potent than the go process (De Jong et al., 1990; 1995; Coxon et al., 2007; Boucher et al., 2007a). The stop process therefore overtakes the go process arriving at the sensory finish-line first (Figure 6.13). Disinhibition of somatosensory cortex (S1) follows, leading to an increase in shock detection rates. Thus, the go process wins the motor race while the stop process wins the sensory race. The sensory decision is implemented before the movement is made even though it occurred after the movement was triggered. This interpretation therefore explains how sensory suppression effects can precede movement. However, if the stop process does not fully inhibit movement as is the case on stop-respond trials, then premotor sensory suppression associated with responding is quickly activated (see stop-respond trials; Figure 6.9, -50 ms to EMG onset). If this explanation is correct it would indicate that the premotor regulation of sensation is dissociable from the premotor decision to act and occurs slightly later. This interpretation predicts a very rapid response of sensory processing to motor decision (see also Chapter 10). In this way, an independent horse-race is achieved while the sensory consequences of going and stopping are both present in rapid sequence.

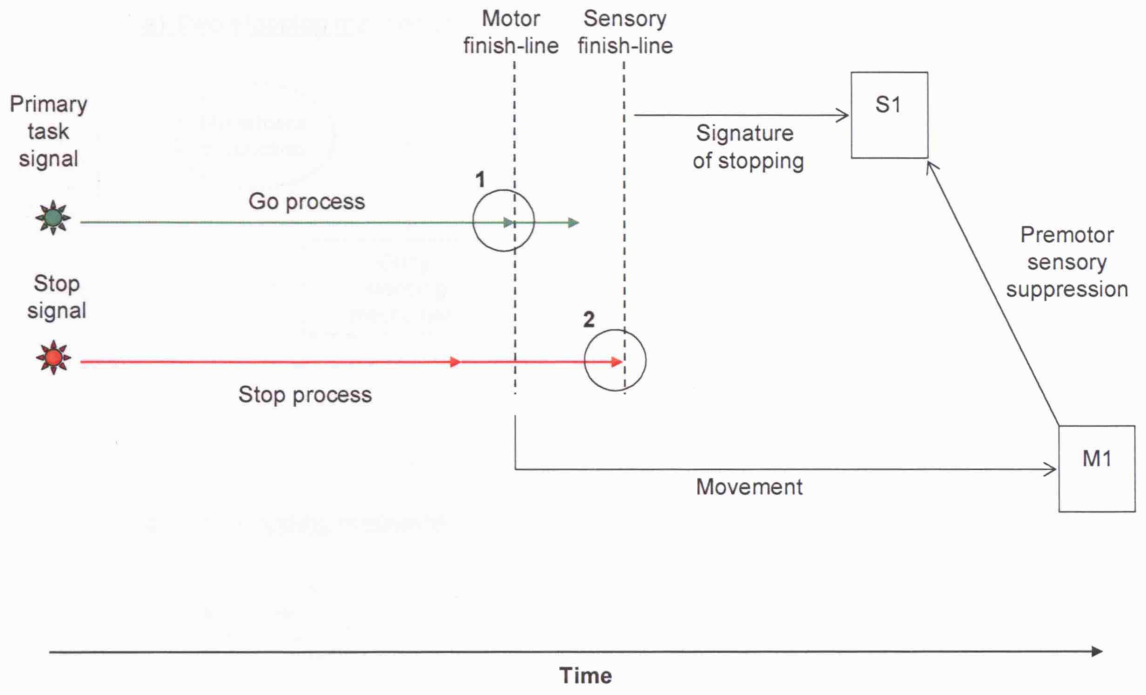

Figure 6.13. Schematic showing possible independent horse-race model of our results. There are two finish-lines, a motor finish-line and a sensory finish-line (vertical dashed lines). On a stop trial, the go (primary task stimulus) signal is presented first, followed on average 96 ms later by the stop signal. Both go and stop processes race towards the motor finish-line. The go process arrives first (circle "1"), triggering the movement via primary motor cortex (M1). The two processes continue to race towards the sensory finish-line. However, the faster and more powerful stop process overtakes the go process arriving at the sensory finish-line first (circle "2"), activating the sensory signature of stopping via somatosensory cortex (S1). Sensory processing is implemented faster than motor processing (as indicated by the shorter distance between the sensory finish-line and S1 than the motor finish-line and M1). Therefore, even though a movement is triggered the sensory signature of stopping is flagged.

6.5.3 One stopping mechanism or two?

There is considerable debate in the literature about the nature of stopping. Two stopping mechanisms of inhibitory motor control have been suggested (Figure 6.14(a); De Jong et al., 1990; 1995), while other researchers have argued for just one (Figure 6.14(b); e.g. van Boxtel et al., 2001).

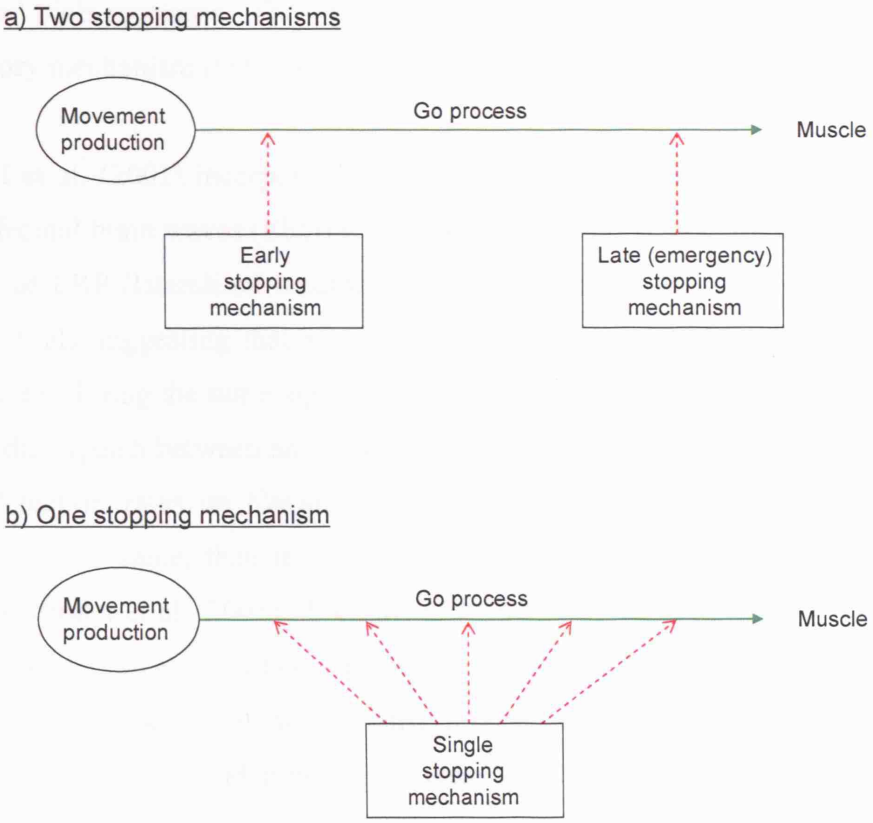

Figure 6.14. Different accounts of stopping. a) Some accounts (De Jong et al., 1990; 1995) propose two separate stopping mechanisms, an early mechanism that can stop movement production and a later "emergency brake" mechanism that can stop a highly activated go process. b) Other models (van Boxtel et al., 2001) propose a single stopping mechanism that can stop the go process at any point along the route from movement production to muscle activation.

De Jong et al. (1990; 1995) postulated that two separate inhibitory mechanisms are involved in the stop process. First, an early mechanism that is relatively slow but can inhibit selectively. Responses that escape the early mechanism and are on their way to the effector muscle can still be intercepted and stopped by a second late mechanism that acts as an "emergency brake", resulting in fast inhibition. Stopping is relatively easy on NoGo trials as the response process has not yet fully developed and muscle activity has not started at the time the NoGo signal is presented. Therefore, it has been hypothesised that NoGo trials involve the early mechanism. However, on stop trials, stopping is relatively difficult as the stop-signal delay is increased and is closer to the moment of response execution; cortical outflow may already have commenced and the probability of

stop-respond trials increases. Thus, it is has been hypothesised that stop trials involve the late inhibitory mechanism (van Boxtel et al., 2001).

Van Boxtel et al. (2001) incorporated NoGo trials into their stop-signal task design and compared frontal brain waves (EEG) in NoGo and stop-signal situations. They found that the pattern of LRP (lateralised readiness potential) responses was similar on NoGo and stop-signal trials suggesting that a *single* mechanism may initiate inhibitory control in both situations. Using the same logic as van Boxtel and colleagues, shock detection could be used to distinguish between an early and a later, more powerful, stopping mechanism. If shock detection rates on NoGo trials (early inhibition) and stop-inhibit trials (late inhibition) are the same, then this would argue in favour of a single motor inhibitory mechanism (Boxtel et al., 2001). If on the other hand, shock detection rates were not the same, then this would suggest two separate mechanisms (De Jong et al., 1990; 1995). In this experiment, shock-timing was adjusted in order to compare movement trials i.e. no-stop-signal and stop-respond trials. A future version of this experiment could adjust shock timing in order to compare shock detection rates on NoGo and stop-inhibit trials so as to investigate if there are one or two stopping mechanisms (De Jong et al., 1990; 1995; van Boxtel et al., 2001). Nevertheless, the current experiment provides some evidence for a single stopping mechanism. Here, the duration of stopping as mathematically estimated using the horse-race model, was found to be 203 ms when subjects managed to successfully inhibit movement on approximately 50% of stop trials. In Experiment 4(a) (Chapter 5) the stopping duration was estimated to be approximately 200 ms when subjects managed to stop successfully on 95% of NoGo trials (errors of commission = 5%). Thus, despite very different probabilities of inhibition, the duration of stopping was very similar suggesting that a single inhibitory mechanism was at play. Other studies report similar stopping durations e.g. De Jong et al. (1995) = 174 ms; De Jong et al. (1990) = 203 ms; van Boxtel et al. (2001) = 174 ms and Kok et al. (2004) = 224 ms. The results are therefore in good agreement with the literature on stopping motor responses and the horse-race model.

In conclusion, using a novel combination of the stop-signal paradigm and the sensory-detection (S-D) task, we observed sensory evidence for the initial activation of a stopping process on trials where the go process wins and a response occurs. The sensory consequences of action suggest that the balance between stopping and going is not just a two-horse race with a winner takes all solution. At least transiently, the sensory system can show a signature characteristic of stopping while the motor system is presumably dominated by the go process. The pattern of sequential sensory activation observed here suggests that stopping and going are not mutually exclusive and independent (cf., Logan et al., 1984). Our data further suggest that the stopping process is activated much earlier and has a longer duration than a current model proposes (Boucher et al., 2007a). When go and stop processes are placed in direct competition against each other, there is a brief time window when the sensory system is in stop mode while the motor system is in go mode.

Here, we combined the sensory-detection task with the stop-signal paradigm. In the next chapter we introduce the "go-signal paradigm", a new task designed to study the motor-sensory interaction when subjects switch from stopping to going.

Chapter 7: Sensory suppression in the "go-signal paradigm"

7.1 Abstract

Here, we combined the sensory-detection task with the "go-signal paradigm", a new task introduced to study the interaction of stop and go processes. Subjects were required to withhold a right index finger movement when a NoGo signal was presented. Occasionally, the NoGo signal was interrupted by a Go signal ("hybrid-Go" trials) and subjects had to initiate a response as quickly as possible. Thus, subjects switched from stopping to going within the same trial. The switch was accompanied by a reaction time cost or "transition delay" of 44 ms relative to Go trials. Normal premovement sensory suppression effects were observed on Go trials. Crucially, premovement sensory suppression on hybrid-Go trials, which had been dismantled to a significant degree following the NoGo signal, did not differ from Go trials by the end of the delay. During the transition delay, the sensory signature of the stop process seems to be completely abandoned and an entirely new sensory response process initiated. As in Chapter 6, evidence for *sequential* sensory activation of the processes for stopping and going was observed. When there is a switch from stopping to going the obsolete stop process is first suppressed, and then the response is initiated.

7.2 Introduction

In Chapter 6 we investigated the sensory consequences of motor inhibition using the *stop-signal paradigm.* In that task, subjects responded to a Go signal though they were occasionally required to withhold their response to the primary task when later presented with a stop signal within the same trial. In the current experiment, we partly reversed these procedures to produce what we call the "*go-signal paradigm*". It is proposed that the go-signal paradigm provides a new way to study the interaction of stop and go processes. In this task, subjects are presented with a NoGo signal cueing them to withhold a response, followed by a Go signal instructing subjects to reinstate the response. Thus, subjects switch from stopping to going within the same trial. These "NoGo-followed-by-Go signal" trials are hereafter labelled "hybrid-Go" trials. Across studies, estimates of stopping suggest it takes between 200 and 250 ms for young adults (e.g. Experiments 4 and 5; De Jong et al., 1990; De Jong et al., 1995; Kok et al., 2004; Logan, 1984; van Boxtel et al., 2001). Therefore, 100 ms after the onset of the NoGo signal, the stop process should already be well underway (Experiments 4; Walsh and Haggard, 2007) and a sudden change of signal instructing subjects to go, should incur a

considerable handicap in the race to initiate a response. Though a response can invariably be made on hybrid-Go trials, it is expected to be delayed relative to Go trials. One reason why this might occur is that there may be a brief interval when the stop process on the first part of the trial (triggered by the NoGo signal) competes with the nascent go process on the second part of the trial (triggered by the Go signal). Because of this competition, subjects may find it more difficult to initiate a response on hybrid-Go trials as reflected by longer reaction times relative to Go trials. This predicted prolongation of reaction time is hereafter called the "transition delay" (Figure 7.1).

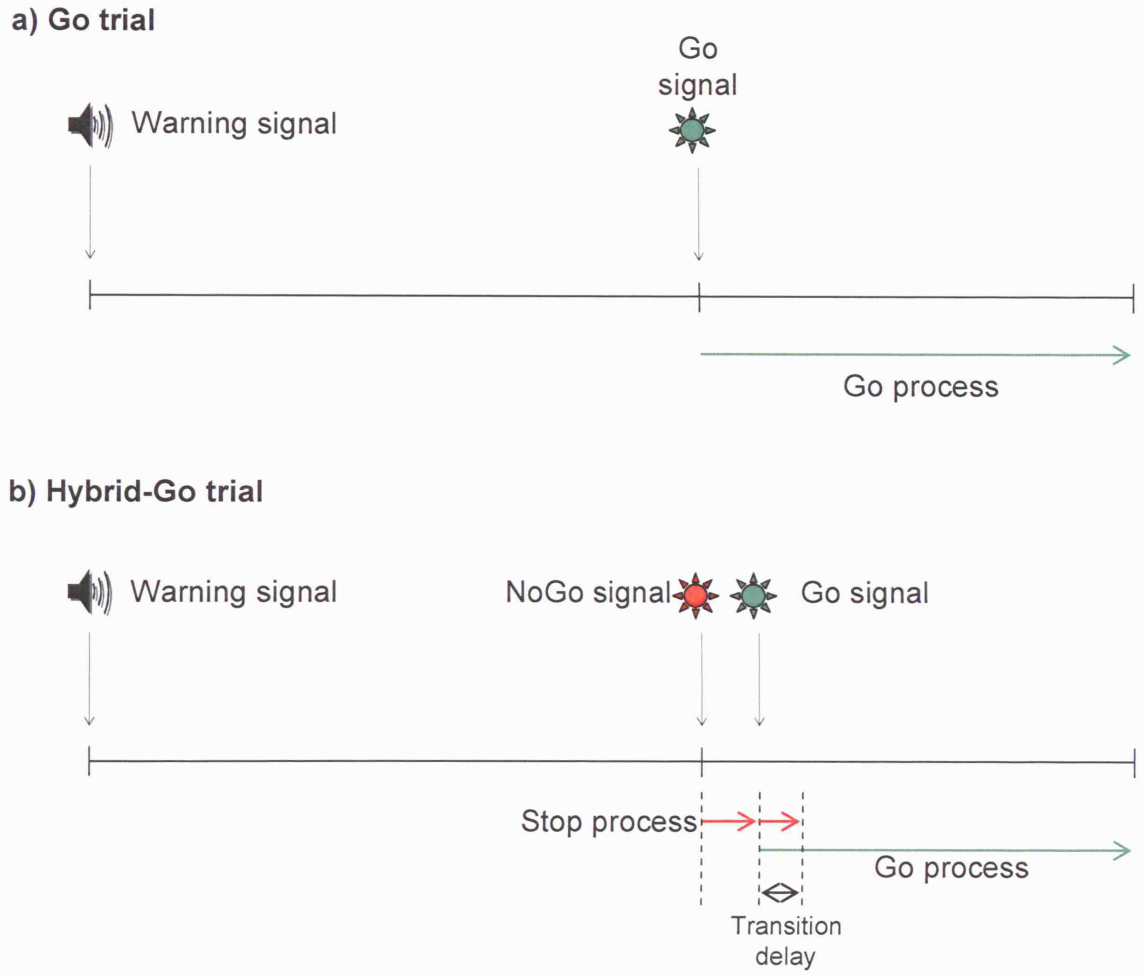

Figure 7.1. Schematic of activation of go and stop processes during Go and hybrid-Go trials. a) On a Go trial, the go process (green arrow) is triggered by the go signal. b) On a hybrid-Go trial, the NoGo signal triggers a stop process (longer red arrow). The stop process may not switch off instantly and may persist (shorter red arrow) for some time after the Go signal. Thus when the Go signal is presented, the persisting activation of the stop process may compete with the developing go process resulting in a prolonged reaction time (transition delay) on hybrid-Go trials relative to Go trials.

It is important to know the duration of the transition delay. Delivering shocks at three different intervals at the end of the transition delay can allow us to directly compare sensory suppression on hybrid-Go trials and Go trials. Using this design, we can establish how long it takes for the sensory system to *switch on* after the stop process has been activated by a NoGo signal for some time. If premovement sensory suppression is fully activated on hybrid-Go trials by the end of the transition delay, then it should not be different from sensory suppression on Go trials where no stop process was present. Alternatively, differences in sensory suppression between Go and hybrid-Go trials would indicate a sensory system that reacts slowly to ongoing modifications in motor behaviour. Specifically, there are a number of different ways whereby the motor and sensory systems might interact. First, the sensory dismantling processes initially activated by the NoGo signal might be too powerful for the go process to overcome and therefore sensory suppression might not switch on at all [Figure 7.2.(a)]. A second possibility is that sensory suppression might activate *fully*, switching the sensory signature from stopping to going rapidly and decisively [Figure 7.2.(b)]. Finally, the go process is sufficiently strong to *partially* neutralise the pre-activated stop process and therefore sensory suppression switches on to a moderate extent only [Figure 7.2.(c)].

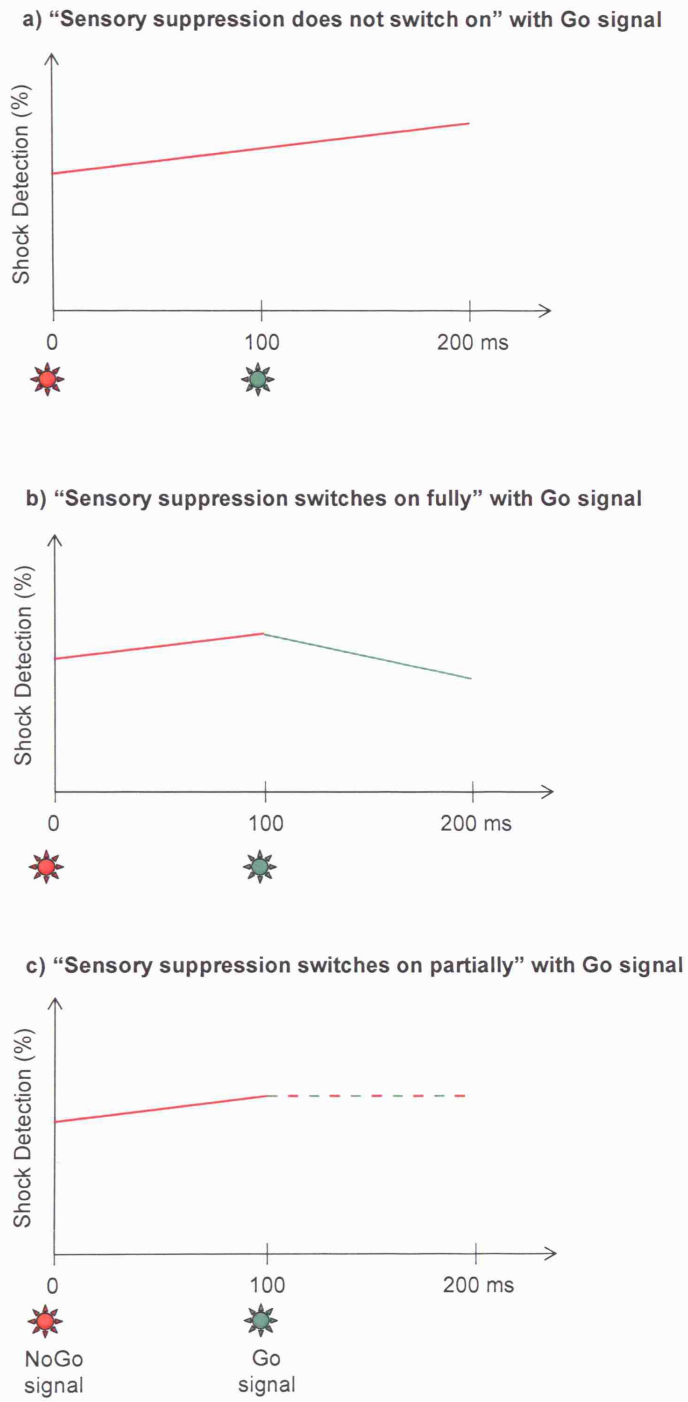

Figure 7.2. Schematic of possible interactions between motor and sensory processes during hybrid-Go trials. Hypotheses include a) Sensory suppression is not activated at all when the Go signal is presented and sensory dismantling dominates. b) Sensory suppression is fully activated by the Go signal causing a rapid switch from dismantling to the classic premovement decrease in shock detection rates or c) sensory suppression is partially activated by the Go signal resulting in a mixture of sensory dismantling and suppression.

Before performing the main experiment it will be necessary to carry out a pilot study in order to calculate the duration of the predicted transition delay. Knowing the duration of the transition delay will allow us to match the delivery of the shock for Go and hybrid-Go trials, thereby facilitating a direct comparison of the sensory processes unfolding with each event. A pilot study and the main Experiment are now presented.

7.3 Pilot Study; Materials and Methods

7.3.1 Subjects

Eight paid subjects took part; 5 were female, 7 were right-handed and the mean age was 23.5 (SD = 4.5) years.

7.3.2 Procedure

A brief acoustic tone marked the onset of the trial. Trials consisted of Go, NoGo, hybrid-Go and catch (no shock stimulus) trials. On Go trials, after 1000 ms a Go signal (a green LED for half the subjects and a red LED for the remainder) was presented for 700 ms. On NoGo trials, a NoGo signal was presented for 700 ms. On hybrid-Go trials, a NoGo signal was presented for 100 ms and then changed colour to a Go signal for the remaining 600 ms of the response window. Shock stimuli were delivered either at the Go or NoGo signal, or after a 100 or 200 ms delay [Figure 7.3(a) and (b)]. On hybrid-Go trials, the shock was delivered either simultaneously with the NoGo signal, simultaneously with the Go signal or 100 ms after the Go signal [Figure 7.3(c)]. Subjects were informed that on some trials the signal might change colour signifying a change from NoGo to Go. In this case they were instructed to move as quickly and as consistently as possible, in exactly the same way as for Go trials. After each trial, subjects reported verbally ('yes'/'no') whether they perceived a shock stimulus. No feedback was given. Trials were self-paced. The experimenter initiated the next trial after an inter-trial interval of at least 1

second. Subjects performed a practice block of 44 trials, followed by 8 experimental blocks consisting of 54 trials. Each block consisted of 36 movement trials; 6 NoGo and 6 hybrid-Go trials. A further 6 catch trials (no shock stimulus) were divided equally between movement and non-movement trials. The order of the trials was randomised. Subjects took a break midway through the experiment and recommenced when ready.

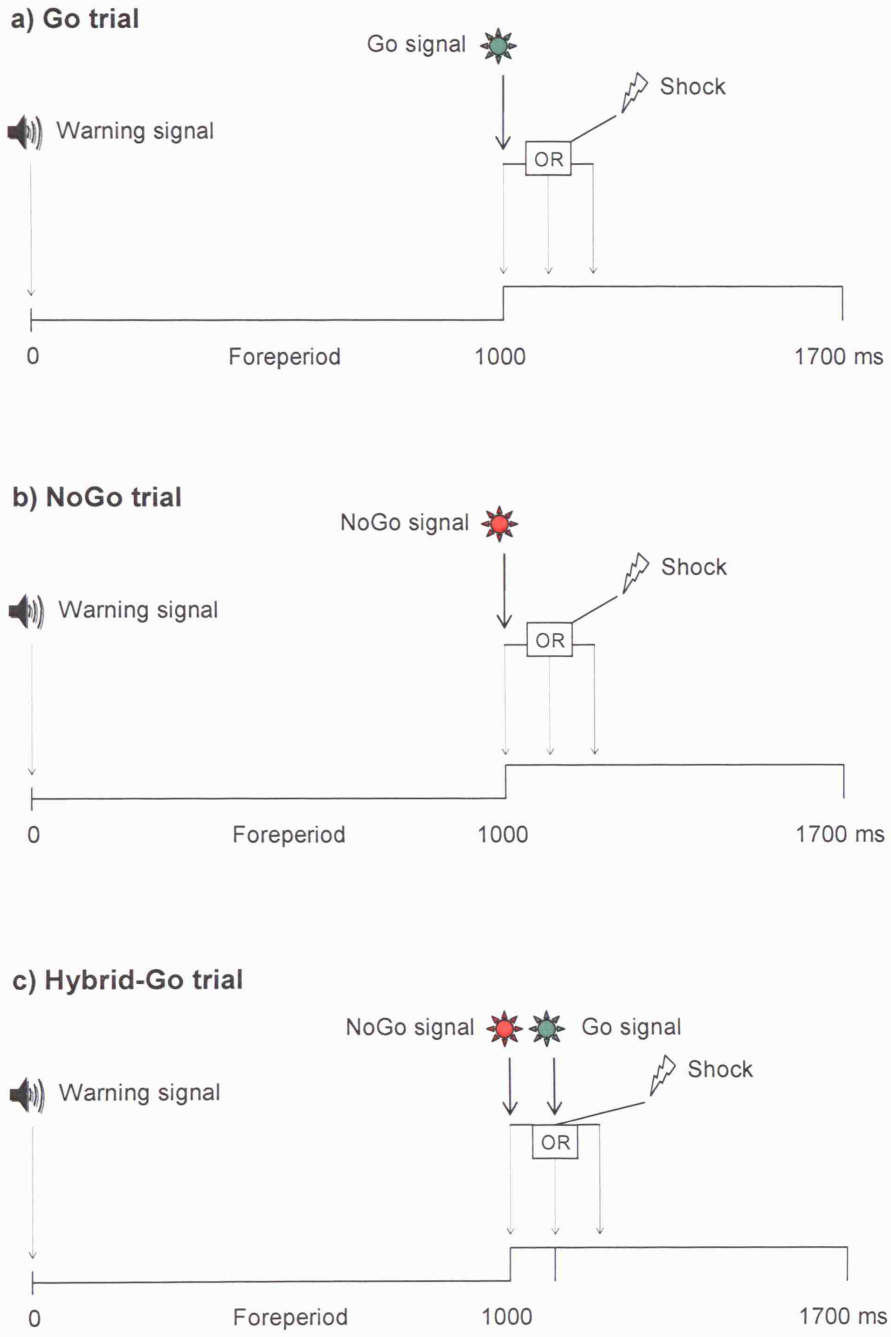

Figure 7.3. Trial design used in the pilot study. An acoustic warning signal marked the start of the trial. After a delay of 1000 ms, the Go or NoGo signal was presented. Subjects waited for and responded to the a) Go signal, but withheld movement following b) the NoGo signal. On hybrid-Go trials, the NoGo signal was presented for 100 ms; the signal then changed colour to signify Go for the remainder of the trial. On all trial types, the shock was delivered simultaneously with the onset of the first signal (1000 ms) or 100 or 200 ms later.

7.4 Pilot Study; Results

Errors of commission (i.e. when a NoGo trial was accompanied by EMG activity and / or overt movement) were 12.5%. The error-rate was unexpectedly high. However, 31% of error trials involved EMG activity with no potentiometer output indicating that subjects managed to halt overt movement at the final stages of response. Shock detection performance on this subset of 15 trials (five subjects) was 26.6%. The high rate of responding on NoGo trials may have been influenced by the presence of the hybrid-Go trials. On these trials, subjects are first signalled to stop (NoGo signal) but then after a delay of 100 ms, the subject is signalled to initiate a response (Go signal) as quickly as possible. This may have lead to a tendency for subjects to prepare to move even when a NoGo signal was presented.

7.4.1 Reaction Times

Reaction times for each subject were trimmed to +/- 2 SD (excluding 2.7% of movement trials). The mean overall RTs for Go and hybrid-Go trials were 251 ms (SD=61 ms) and 386 ms (SD=89 ms) respectively, yielding a difference of 135 ms. This difference was significant; $t(7)=19.296$; $p<0.0001$. This difference in RT equates to 35 ms when the hybrid-Go trial mean reaction time is locked to the Go signal (Figure 7.4).

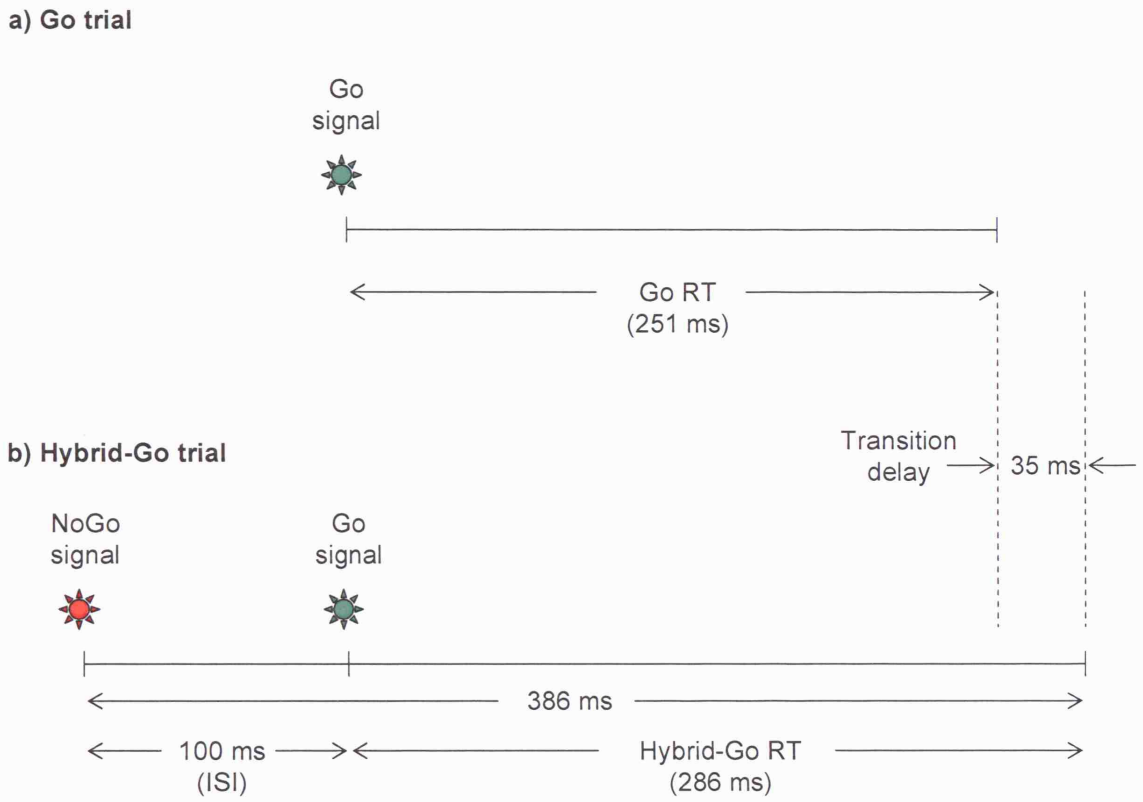

Figure 7.4. Pilot study. Schematic of a) Go trial RT and b) hybrid-Go trial RT. The transition delay (35 ms) was estimated by subtracting Go trial RT (251 ms) from the response-locked hybrid-Go trial RT (286 ms).

Thus, hybrid-Go trials involve either a switch, a loss of preparation or a mixture of both, which is expressed as a delay of 35 ms. This difference of 35 ms is the key information from this pilot study and will be used to match the timing of shock stimuli for Go and hybrid-Go trials in the main Experiment.

7.4.2 Shock detection

Shock detection rates for the pilot experiment were analysed with a repeated measures 3 x 3 ANOVA for the factors trial type (Go, NoGo and hybrid-Go) and stimulus timing (0, 100 or 200 ms after the signal). Table 7.1 shows shock detection rates for each trial type. There was no significant main effect of trial type $F(2,14)=0.919$; $p=0.418$. The main

effect of stimulus timing was also not significant $F(2,14)=0.962$; $p=0.395$. The interaction between trial type and stimulus timing approached but did not reach significance $F(4,28)=3.157$; $p=0.061$.

Table 7.1. The mean percentage of shock stimuli detected (+/- SD) for NoGo, Go and hybrid-Go trial types in the pilot study. On all trial types, the shock was delivered simultaneously with the onset of the first signal or 100 or 200 ms later (see also Figure 7.3).

Time of Shock after Signal (ms)	Trial Type		
	NoGo	Go	Hybrid-Go
0	46 (19)	49 (9)	43 (10)
100	49 (19)	45 (11)	50 (19)
200	44 (18)	32 (17)	50 (8)

7.5 Main Experiment; Materials and Methods

7.5.1 Subjects

Eleven paid subjects took part. Two subjects were excluded because of unstable detection rates (+/-15% of pre-experiment levels). Data from the remaining 9 subjects were included in the final analysis; Seven were female, 7 were right-handed and the mean age was 25.0 (SD=8.8) years.

7.5.2 Procedure

The procedure is identical to the pilot study with the exception of the timing of the shock on hybrid-Go trials (Figure 7.5). On hybrid-Go trials, the timing of the shock was response-locked (based on the pilot study) and therefore delivered at 35, 135 or 235 ms after the Go signal [Figure 7.5(c)]. The aim was to deliver the shock relative to the onset of EMG activity in an equivalent way across all movement (i.e. Go and hybrid-Go) trials in order to be able to compare sensory suppression effects.

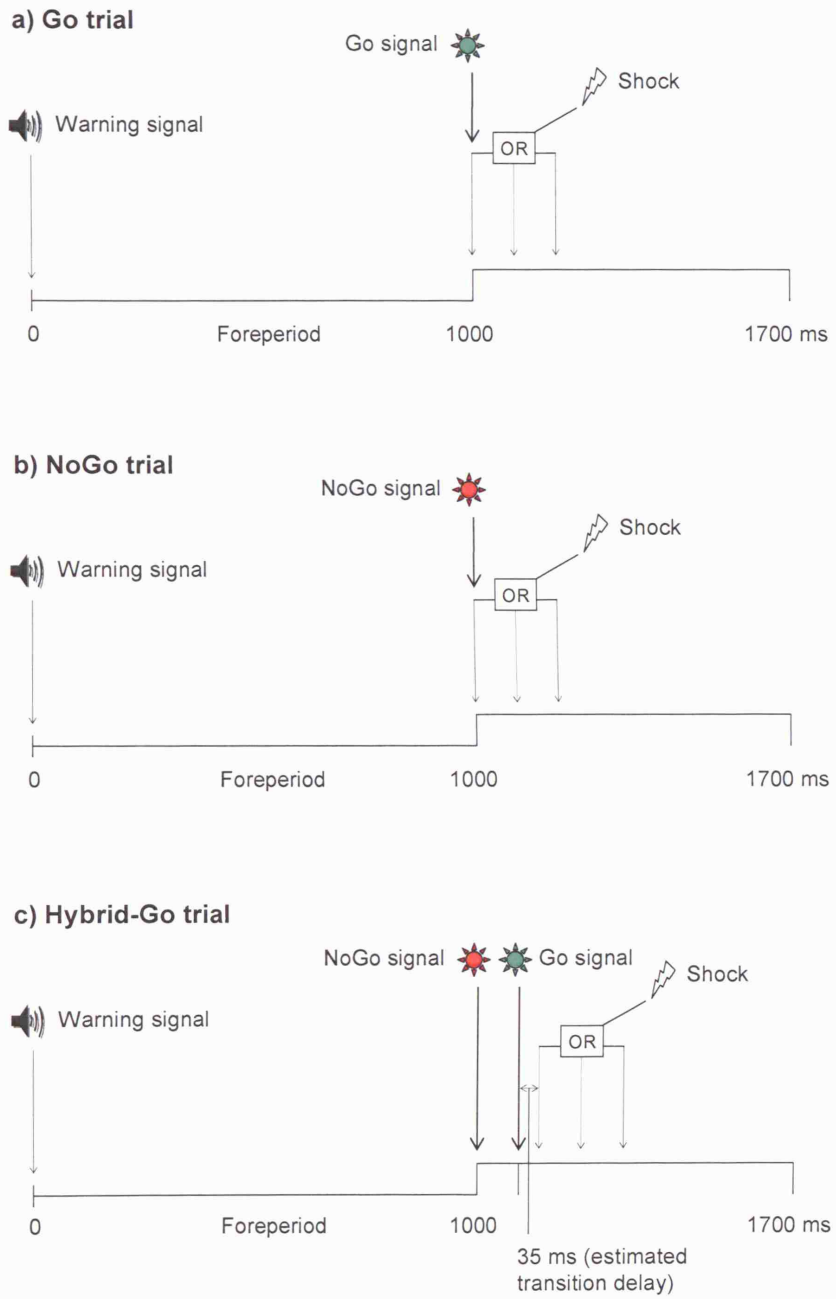

Figure 7.5. Trial design used in the main Experiment. An acoustic warning signal marked the start of the trial. After a delay of 1000 ms, the Go or NoGo signal was presented. Subjects waited for and responded to the a) Go signal, but withheld movement following b) the NoGo signal. On Go and NoGo trial types, the shock was delivered simultaneously with the onset of the first signal (1000 ms) or 100 or 200 ms later. c) On hybrid-Go trials, the NoGo signal was presented for 100 ms; the signal then changed colour to signify Go for the remainder of the trial. On hybrid-Go trials, the shock timing was response-locked (based on the estimated transition delay from the pilot study) and therefore delivered 35, 135 or 235 ms after the Go signal.

As before, subjects performed a practice block of 44 trials, followed by 8 experimental blocks consisting of 54 trials. Each block consisted of 36 Go trials (12 each with the shock at 0, 100 and 200 ms after the Go signal); 6 NoGo trials (2 each with the shock at 0, 100 and 200 ms after the NoGo signal) and 6 hybrid-Go trials (2 each with the shock at 35, 135 and 235 ms locked to the Go signal). A further 6 catch trials (no shock stimulus) were divided equally between movement and non-movement trials. The order of the trials was randomised.

7.6 Main Experiment; Results

On catch trials, where no shock stimulus was delivered, 0.7% false positive detections were recorded. A 3 x 1 ANOVA found no difference in false positive detection rates between movement and non-movement trials for the various trial types; $F(2,16)=2.800$; $p=0.103$ (see Table 7.2).

Table 7.2. The number of false positive catch trials pooled across subjects for Go, hybrid-Go and NoGo trials.

Trial Type	
Go	3
Hybrid-Go	1
NoGo	0

Errors of omission (i.e. movement trials without movement during the response window) occurred on 1.8% of movement trials. These trials were excluded when measuring the effects of sensory suppression.

Errors of commission (i.e. when a non-movement trial was accompanied by EMG activity) on NoGo trials were 11.1%. The error-rate is similar to the pilot study (12.5%) and confirms that subjects tended to prepare to go even when a NoGo signal was presented. A small subset of error trials (N=14) involved EMG activity with no overt movement. Shock detection rates on these latter trials were 29%.

7.6.1 Stability across time of perceptual performance at rest

The pre- and post-experiment staircases showed similar shock intensity thresholds (mean pulse-widths=25.3 and 24.6 μs respectively); $t(8)=0.298$; $p=0.773$.

7.6.2 Reaction Times

Reaction times for each subject were trimmed to +/- 2 SD (excluding 3.8% of all movement trials). On 97.9% of movement trials the shock stimulus was delivered prior to the onset of EMG activity and the remaining 2.1% of trials were discarded as premotor sensory suppression is of primary interest here. The mean overall RTs for Go and hybrid-Go trials (locked to the Go signal) were 286 ms (SD=62 ms) and 330 ms (SD=78 ms) respectively. This difference was significant $t(8)=10.980$; $p<0.0001$. The delay on hybrid-Go trials caused by the transition from Go to NoGo (44 ms) for each subject was computed by subtracting the mean Go RT (286 ms) from the mean response-locked hybrid-Go RT (330 ms; Figure 7.6). The duration of the transition delay (44 ms) was similar to the value estimated in the pilot study (35 ms). In order to compare sensory suppression rates between Go and hybrid-Go trials, the timing of the shock stimulus relative to EMG onset must be similar across both sets of trials as the depth of sensory suppression is a function of the time of the stimulus relative to the onset of the movement. The mean shock – EMG onset intervals for Go and hybrid-Go trials were 186 ms (SD=98 ms) and 192 ms (SD=111 ms) respectively. These intervals did not differ significantly

t(8)=0.515; p=0.620 showing that our adjustment of the shock timing for each trial type based on the pilot study data was successful.

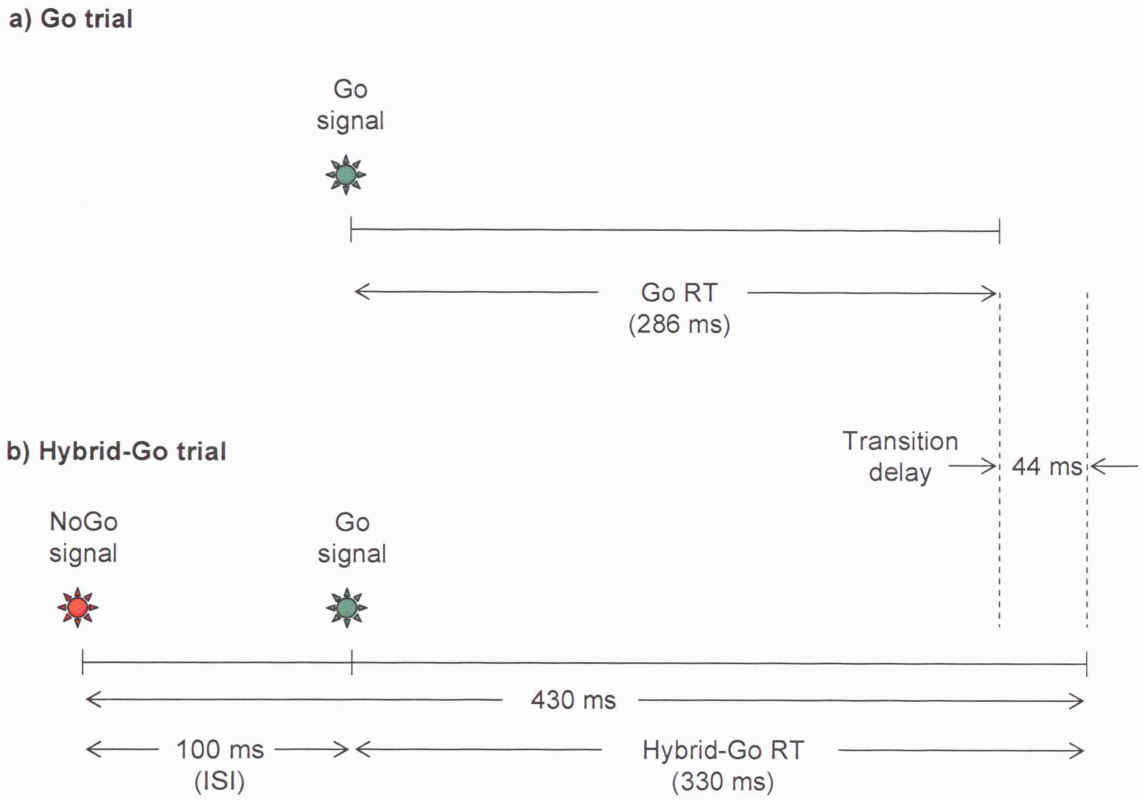

Figure 7.6. Main Experiment. Schematic of a) Go trial RT and b) hybrid-Go trial RT. The transition delay (44 ms) was estimated by subtracting Go trial RT (286 ms) from the response-locked hybrid-Go trial RT (330 ms).

7.6.3 Shock detection

Figure 7.7 shows shock detection rates for NoGo, Go and hybrid-Go trial types. Note that the three bars on the left (NoGo trials) show the dismantling effect with a recovery from sensory suppression with time after the NoGo signal. The three bars in the centre (Go trials) show classic sensory suppression with a reduction in shock detection rates just prior to EMG onset. Importantly, note the similarity in the pattern of the Go trials (three bars in centre) and the hybrid-Go trials (three bars on right). The data were analysed with

a repeated measures 3 x 3 ANOVA for the factors trial type (Go, NoGo and hybrid-Go) and shock timing (0, 100 or 200 ms and 35, 135 and 235 after the Go signal). There was a significant main effect of trial type $F(2,16)=6.399$; $p=0.022$. The main effect of shock timing approached but did not reach significance $F(2,16)=3.461$; $p=0.066$. Interestingly, inspection of Figure 7.7 shows that this trend is in the predicted decreasing direction of the familiar sensory suppression curve for Go and for hybrid-Go trials. As a result of this difference, the interaction between trial type and stimulus timing was significant $F(4,32)=7.297$; $p=0.003$.

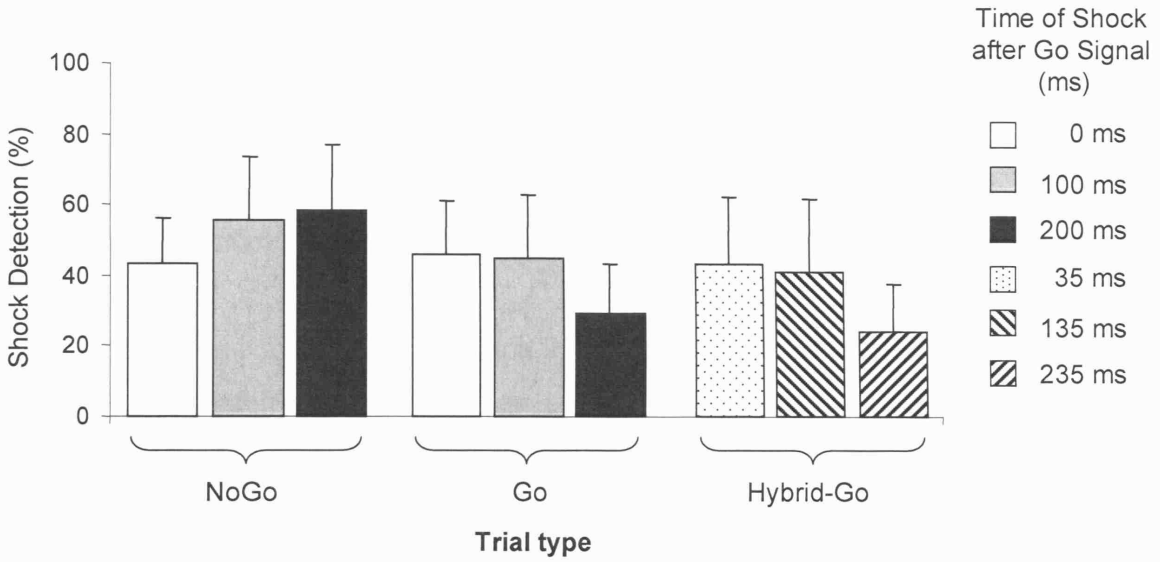

Figure 7.7. The mean percentage of shock stimuli detected (+/- SD) for Go, NoGo and hybrid-Go trial types. On Go and NoGo trials, the shock stimulus was delivered at 0, 100 or 200 ms after the Go or NoGo signal. On hybrid-Go trials, the shock timing was response-locked (based on the pilot study) and therefore delivered at 35, 135 or 235 ms after the Go signal.

A 2 x 3 ANOVA was used to explore movement trials for the factors trial type (Go and hybrid-Go) and shock timing (0, 100 or 200 ms and 35, 135 and 235 after the Go signal). There was no main effect of trial type $F(1,8)=2.090$; $p=0.186$. The main effect of timing was significant $F(2,16)=9.216$; $p=0.003$. However, the interaction was not significant; $F(2,16)=0.054$; $p=0.919$. Crucially, the effects of sensory suppression did not differ for Go and hybrid-Go trial types. Follow-up t tests were used to explore when sensory

suppression first commenced. There was no difference between the early and middle shock-timing $t(8)=0.322$; $p=0.755$. However, the difference between the middle and late shock-timing was significant $t(8)=4.196$; $p=0.003$.

In order to explore if dismantling (i.e. recovery from sensory suppression) took place on NoGo trials a 1 x 3 ANOVA was carried out on NoGo trials only, comparing different levels of shock timing (0, 100 or 200 ms after the NoGo signal). There was a main effect of timing $F(2,16)=4.174$; $p=0.047$. Follow-up t tests (one-tailed) revealed a significant increase in shock detection rates from 0 to 100 ms; $t(8)=3.011$; $p=0.009$. Therefore, significant dismantling was observed to take place in the first 100 ms after the NoGo signal. The rate of recovery from sensory suppression for the subsequent step from 100 to 200 ms was not significant; $t(8)=0.405$; $p=0.348$.

7.6.4 Time-dependent changes in the detection of stimuli applied to the moving digit

To study the time-course of any variation in performance during movement, the data were pooled from all subjects, trials were grouped into 50 ms bins relative to EMG onset and the graph plotted (Figure 7.8). The classic sensory suppression curve was revealed for both Go and hybrid-Go trials (Williams et al., 1998). Weak cutaneous shocks were less likely to be detected just prior to the onset of EMG activity and movement.

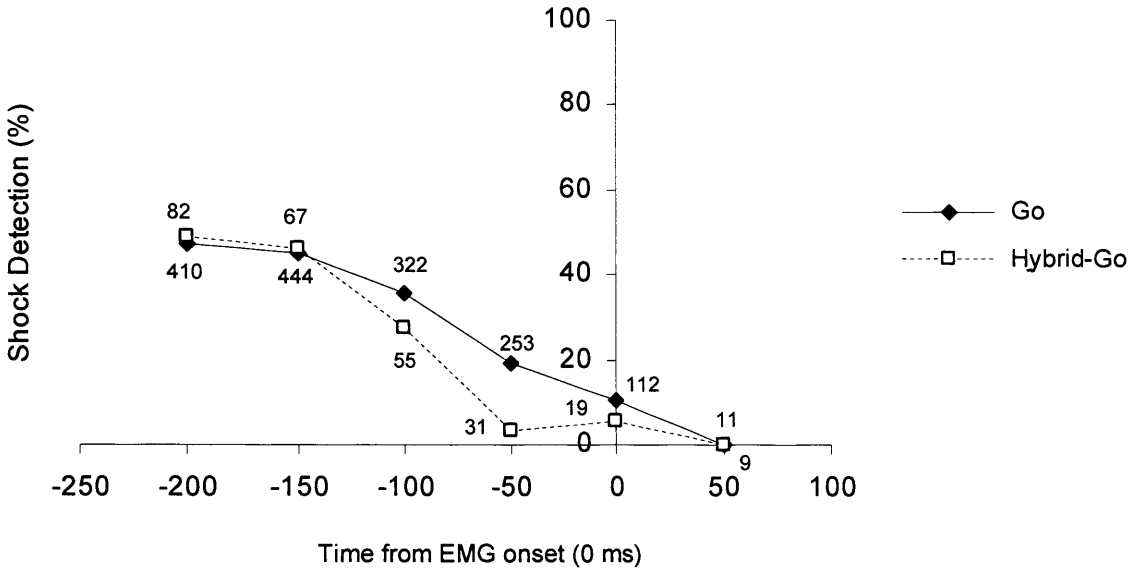

Figure 7.8. Effects of index finger movement on the detection of fixed intensity stimuli applied to the moving finger for Go and hybrid-Go trial types. Detection performance over time is plotted relative to the onset of EMG (0 ms); a negative number indicates that the shock precedes EMG onset. The values next to the data points refer to the number of trials represented at that data point.

A 2 x 6 ANOVA was performed for the factors trial type (Go or hybrid-Go) by time-bin (-200, -150, -100, -50, 0 and 50 ms relative to EMG onset). There was a main effect of trial $F(1,8)=24.247$; $p=0.001$ and a main effect of time-bin $F(5,40)=24.395$; $p<0.001$. However, the trial by time-bin interaction was not significant $F(5,40)=2.373$; $p=0.116$. Note that in Figure 7.8 the two curves diverge for the two time-bins, 100 ms and 50 ms, prior to EMG onset. Exploratory t tests were carried out on this divergence (see Table 7.3).

Table 7.3. Follow-up t tests (p values are two-tailed) comparing shock detection performance for Go and hybrid-Go trial types for each time-bin. Time-bins are relative to onset of EMG (0 ms).

Time-bin (ms)	p value (see also Figure 7.8)
200	0.131
150	0.772
100	0.042
50	0.017
0	0.145

Shock detection performance was significantly poorer in hybrid-Go than Go trials for the two time-bins, 100 ms and 50 ms, prior to EMG onset. It is not clear why premovement sensory suppression should be deeper on hybrid-Go trials than Go trials just prior to movement. During hybrid-Go trials, subjects switch from one process (stopping) to another (going). One possibility is that the sensory system may temporarily overshoot as it adjusts to the sudden demand on motor output. However, this interpretation must be treated with great caution as the number of hybrid-Go trials in each time-bin is very low (see Figure 7.8).

7.7. Discussion

Here, we asked how long does it take for sensory suppression to switch on after inhibitory processes have been activated for a duration of 100 ms. Sensory suppression, which had been dismantled to a significant extent following the NoGo signal on hybrid-Go trials, had been successfully reactivated by the end of the "transition delay". There was no sensory evidence of the stop process on hybrid-Go trials. Indeed, sensory suppression effects on hybrid-Go trials were similar to Go trials where no dismantling had taken place. During the transition delay on hybrid-Go trials, the sensory manifestation of the stop process seems to be completely abandoned and an entirely new sensory response process started. This implies that both the coupling and decoupling of sensorimotor links

in preparation for the new task are fully complete by the end of the delay. Sensory suppression on hybrid-Go trials is switched on rapidly, despite the presence of inhibitory processes for the initial part of the trial. The results are therefore consistent with the "sensory-suppression-switches-on-fully" hypothesis [Figure 7.2.(b)].

The duration of stopping typically takes 200 ms in young adults (Logan, 1984). Therefore, the stopping process should be well underway after 100 ms. However, it could be argued that perhaps the stop process was very weak or was not even activated on hybrid-Go trials at the time the Go signal was presented. In this case, it would be very easy for a burgeoning go process to overcome the hardly activated stop process. This would explain why normal sensory suppression effects were observed after the Go signal on hybrid-Go trials. However, our results suggest that this was not the case. An inspection of NoGo trials (see Figure 7.7) indicates that significant sensory dismantling took place in the first 100 ms after the NoGo signal. Therefore, inhibitory processes were activated by the time the Go signal was presented on hybrid-Go trials. Yet, even though the stop process was present at the start of the hybrid-Go trial, there was no sensory evidence of stopping after the end of the delay, 35 ms later.

During a hybrid-Go trial one can conceive of two ongoing processes – a stop process that is triggered with the onset of the NoGo signal as well as a go process triggered by the onset of the Go signal. When the Go signal is presented the stop process may not be instantly suppressed. It may take time for stop process activation to dissipate. Persisting stop process activation may temporarily compete with the developing go process activated by the Go signal (Figure 7.9). Our results suggest a *sequential* generation of the processes for stopping and going. This sequential pattern of processes is reminiscent of the pattern observed in the stop-signal paradigm (Chapter 6). In the stop-signal paradigm, the sensory evidence suggested that when go and stop processes are placed in direct competition against each other, a brief pattern of sequential ("stop-then-go") sensory activation is observed, where the sensory system is in stop mode while the motor system is in go mode. In the current experiment, we found a similar pattern of sequential sensory

activation on hybrid-Go trials. First, the defunct stop process is suppressed in 35 ms or less. Second, the response process is initiated.

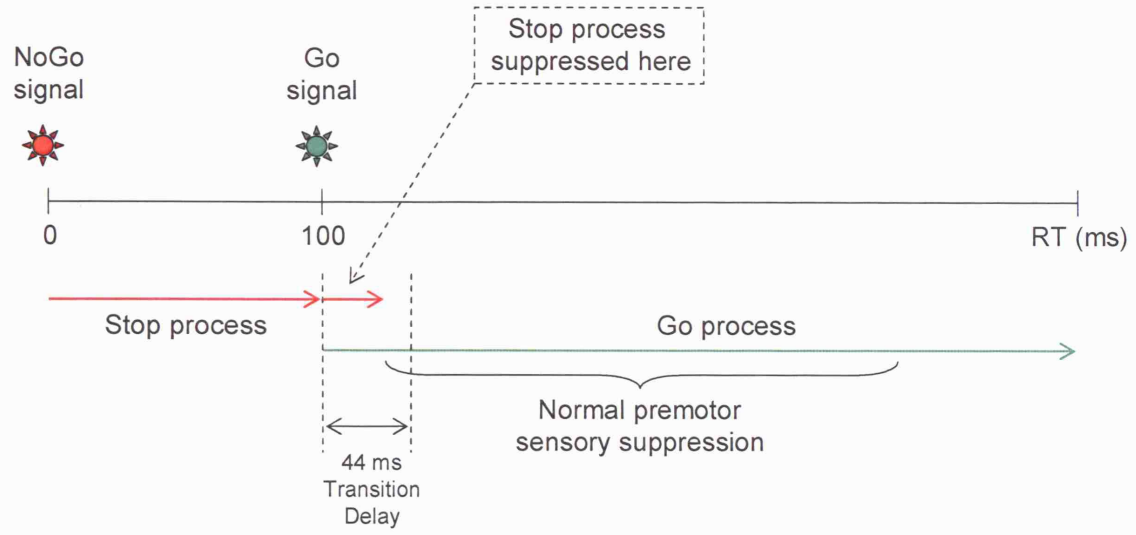

Figure 7.9. Schematic of activation of go and stop processes during a hybrid-Go trial. The NoGo signal triggers a stop process (long red arrow) which can persist (short red arrow) resulting in a prolongation of reaction time i.e. the transition delay. Normal premotor sensory suppression was measured from 35 to 235 ms after the Go signal (indicated by horizontal bracket) which suggests that the stop process was suppressed in the first 35 ms of the transition delay.

On hybrid-Go trials, the predicted short transition delay (44 ms) in reaction time relative to Go trials was observed. This value is similar to the 35 ms delay latency calculated in the pilot study, suggesting that the transition delay is a robust phenomenon. The delay is interpreted as evidence of the competition between persisting activation from the stop process triggered by the NoGo signal at the start of the trial and the developing activation of the go process triggered by the Go signal. Some cognitive control is required in order to overcome the stop process activated by the NoGo signal and to initiate the sensory suppression associated with the response. This process takes measurable time but is surprisingly quick; modulation of somatosensory cortex (S1) can begin within 35 ms of a Go signal. Psychologists often think of modulation as a slow contextual process. Our

result suggests that modulation, in the context of motor-sensory interaction at least, can be rapid and signal-driven.

In the current Experiment, we modified the stop-signal paradigm to create the "go-signal paradigm" where the *Go* process is under experimental control. Most studies interrupt the go process with the stop process using the stop-signal paradigm (e.g. Logan and Cowan 1984; van den Wildenberg, van Boxtel and van der Molen, 2003). Here, we interrupted the stop process with the go process. By "reversing" existing procedures, we introduce a new way to study motor-sensory interactions. Our results suggest that the stop process can be interrupted quite easily in much the same way as the go process can be (Chapter 6; De Jong et al., 1990; Logan and Cowan, 1984). Future research could systematically investigate the relationship between the stop and go processes by manipulating the interval between the onset of the NoGo and the Go signal on hybrid-Go trials (the "*go-signal delay*" or "GSD" see Figure 7.10). The central question would be if the duration of the transition delay and sensory processing vary as a function of the go-signal delay (GSD). Stopping will presumably be stronger at shorter GSDs as persisting stop process activation may still be present and the go process is nascent. Thus, as the GSD becomes shorter, the stop process may become stronger and the go process weaker resulting in less competition. This in turn may slow down reaction times and weaken premotor sensory suppression. An absence of the sensory signature of stopping at short GSDs would confirm that the stop process is indeed completely suppressed during the transition delay. In the converse situation where the GSD is longer, the stop process may be weaker than the rapidly developing go process. In this case, it is predicted that a response would be initiated faster and accompanied by stronger premotor sensory suppression.

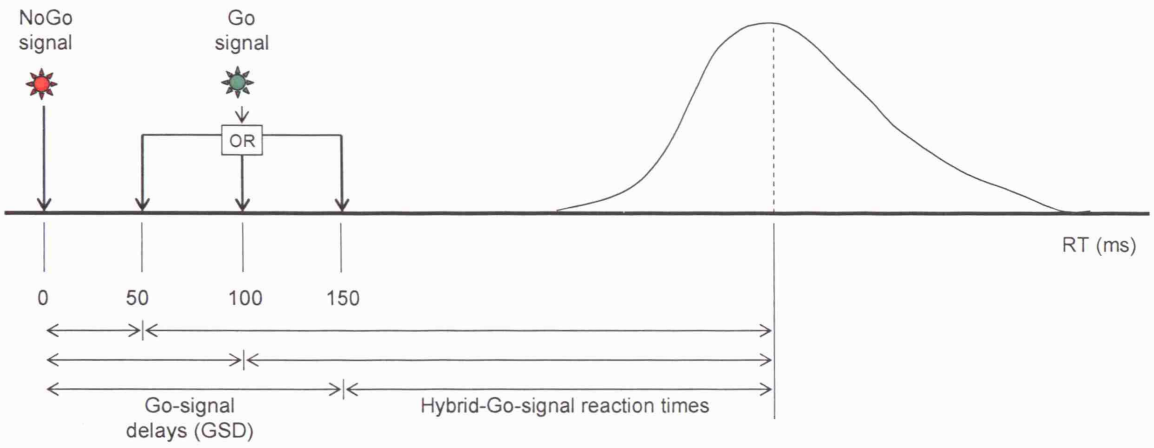

Figure 7.10. The go-signal paradigm. The curve depicts an RT distribution for Go trials, representing the finishing time of the response processes. The hybrid-Go-signal reaction time (GSRT) is calculated by subtracting the go-signal delay from the mean hybrid-Go RT.

An interesting question raised by the current experiment is what happens during the transition delay. Here, the delay was interpreted as evidence of the competition between persisting activation from the stop process and the developing activation of the go process. During the delay, sensory processes are somehow reconfigured as subjects mentally "change-gear" from stopping to going. Sensory suppression was not measured during the transition delay in the current Experiment. Future research could deliver shocks during the delay in order to map the sensory transition from stopping to going (Figure 7.11).

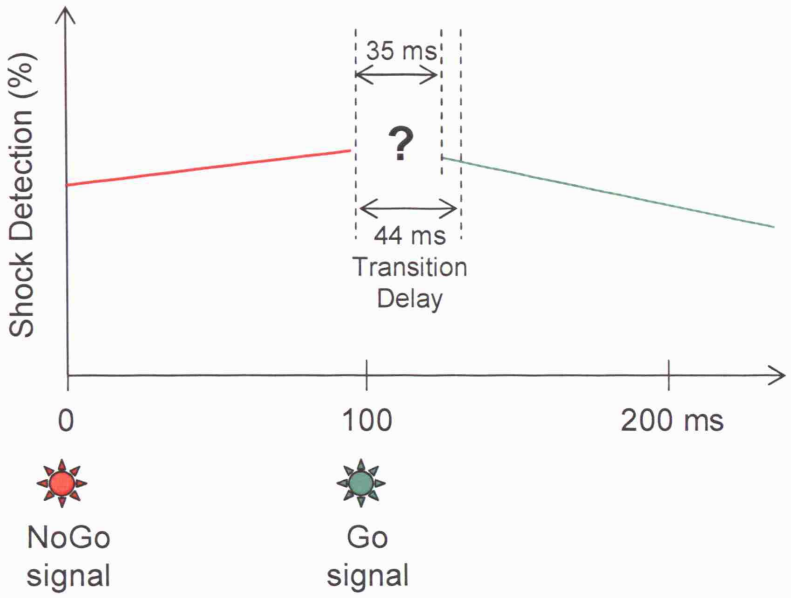

Figure 7.11. Schematic of interaction between motor and sensory processes observed during hybrid-Go trials. Sensory dismantling in the first 100 ms after the NoGo signal was confirmed on NoGo trials (red line). Premotor sensory suppression was fully activated (green line) by the end of the transition delay (dashed lines). However, the internal structure of the first 35 ms of the transition delay is not known.

The rate of errors of commission (i.e. EMG activity and / or movement on NoGo trials) was quite high (11%). This relatively high rate of errors of commission was also observed in the pilot study. Errors of commission are usually interpreted as a measurement of insufficient response inhibition. However, the high rate of errors found on the current task may reflect a by-product of the inclusion of the hybrid-Go trials. On half of all NoGo trials, subjects must overcome inhibition and suddenly activate a response upon arrival of a Go signal. The present data indicate that the occasional presence of the hybrid signal may have interfered with NoGo performance. Subjects may have strategically increased their readiness to respond or adjusted their motor activation level (Chapter 4; Näätänen, 1970; Näätänen and Niemi, 1981) because they ultimately expected a Go signal. This strategy would have produced more errors of commission. However subjects did not ignore the NoGo signal as the familiar NoGo dismantling of sensory suppression was present (Figure 7.7).

A methodological issue in the previous experiment (Experiment 5) was that the task involved a change in the visual signal within a trial. It is possible that this change may have had an arousing effect on subjects, thereby resulting in the upward inflection in shock detection observed on stop-respond trials (see Figure 6.9). Previously, Experiment 5 could not rule out this possibility. Here, we provide evidence that this was not the case as opposite sensory suppression effects were observed in the current task which also involved a signal change within a trial. The observed variations in sensory suppression therefore do not reflect arousal but instead seem to reflect the specific cognitive demands of each task.

In conclusion, using the new "go-signal paradigm", we found further evidence for the sequential sensory activation of stop and go processes. When there is a switch from stopping to going, the obsolete stop process is first suppressed and then the response is initiated.

The Go/NoGo task and the stop paradigm are key tools for investigating what happens when a prepared movement is cancelled. However, movements are not always cancelled. Sometimes it is necessary to replace one movement with another in order to interact effectively with our environment. In the "Posner paradigm" (Posner et al., 1980), subjects must switch from a prepared action to a new action. In the next chapter we ask how sensory processing is affected when one hand is primed for movement, but then the other hand is unexpectedly recruited.

Chapter 8: Sensory suppression in the "Posner paradigm"

8.1 Abstract

Here, we combined the sensory-detection task with the "Posner paradigm" in order to test if movement expectancy influences sensory suppression. Subjects were required to abduct either their right or left index finger as quickly as possible after the presentation of a discriminative visual stimulus and to detect weak shocks that were delivered to the right index finger on some trials. There was either a low (20%), medium (50%) or high (80%) likelihood of a right finger movement within a block. Subjects produced slower reaction times to unexpected movements as predicted. When expectancy was low the right hand was slower than the left hand; reaction times for both hands were similar for the medium and high expectancies. Also as predicted, shock detection performance prior to left and right hand movements was worse for the right (shocked) hand than the left (non-shocked) hand for the low and medium expectancy conditions, replicating the finding that body parts remote to the point of stimulation do not gate transmission of the afferent input to the same extent (Experiment 2; Williams et al., 1998). Interestingly, we found similar attenuation of sensory inputs from the right hand when subjects expected to move the right hand but in fact moved the left in response to rare left signals. It is concluded that expectancy contributes to sensory suppression.

8.2 Introduction

The processes involved in advance movement preparation and action anticipation are of key interest since these processes play an important role in the optimisation of motor performance (Requin et al., 1991). One of the methods used to study the ability to plan and change behaviour according to task requirements is the "Posner paradigm" (Posner et al., 1980). Posner and colleagues provided subjects with a precue as to whether a stimulus would occur to the left or the right of fixation. When the stimulus occurred in the expected condition (80% probability), subjects' reaction times were faster than following the medium cue (50% probability), whereas when the stimulus occurred at an unexpected position (20% probability), they were slower. Larish and Stelmach (1982) instructed subjects to generate rapid arm movements in response to the presentation of a target, which was preceded by a precue. The precue provided correct information about the upcoming target presentation in 80% of the trials, whereas in 20% of the trials, the precue information was incorrect. A higher probability of valid precue information (80%) resulted in a stronger predisposition to expect the corresponding target. If

however, the precue was invalid i.e. was followed by another non-corresponding target, then the prepared plans of movement need to be discarded and new ones need to be prepared online and executed depending on the actual location. Thus, for arm movements, an effect of faster reaction times in valid trials relative to invalid trials was found, replicating Posner et al. (1980) and suggesting that advance information invokes planning processes prior to response initiation (Larish and Stelmach, 1982). These behavioural effects presumably reflect a time-consuming re-programming of the falsely prepared response parameters in the invalid trials (Leuthold, 2003). This RT difference has been explained in terms of spatial attention to the expected location facilitating sensory processing at that location (Posner et al., 1980) or stronger motor preparation of the response associated with the expected stimulus. More recently, the "premotor theory of attention" (Rizzolatti et al., 1994) has proposed that attention and motor preparation are in fact a single process.

Physiological experiments suggest that the precue can trigger the activation of the motor cortex several hundred milliseconds before the movement is executed (e.g. Eimer, 1998) giving rise to a lateralised readiness potential (LRP). The LRP is an electrophysiological indicator of unimanual response activation in primary motor cortex, reflected by an enhanced negativity over the motor cortex contralateral to the side of the activated response (Eimer and Coles, 2003; Eimer, 1998). It has also been shown that unimanual preparation effects can be generated endogenously, without precues, simply by subjects learning the probabilities of different stimuli (van der Stigchel and Theeuwes, 2007). When subjects prepare left-hand or right-hand responses prior to the onset of the imperative stimulus, the cortical motor pathway is activated in readiness for the response. One potential consequence of this motor activation may be suppression of the sensory system. Thus, preparation can potentially affect sensory processing in two opposite ways. First, attentional processes can result in enhanced performance when detecting tactile stimuli presented to the attended hand. Alternatively, when subjects prepare a response, the motor cortex is activated in readiness for the response which may result in suppression of the sensory system.

In the present study, subjects generated rapid left or right finger abductions in response to a discriminative visual cue. In each block there was either a low (20%), medium (50%) or high (80%) likelihood of a right hand movement. No explicit information about movement probability was given to the subjects and there was no precue. Faster reaction times were predicted for expected movements, and slower reaction times for unexpected movements, relative to a condition where both movements were equally likely.

The main aim of the current experiment was to explore the effects of expectancy, if any, on sensory suppression. It has been shown that the inhibitory influence of sensory suppression is largely restricted to the moving digit, (Experiment 2; Williams et al., 1998). Therefore, it was predicted that shock detection performance would be worse for the right (shock stimulus) hand than for the left (no stimulus) hand. However, the effects of expectancy might work in either direction. On the one hand, sensory processing might be facilitated as a result of greater attention to the right hand. Alternatively, the greater motor preparation for the expected response could cause sensory suppression as the probability of a right finger movement is increased. Thus, two competing hypotheses are presented, the "sensory facilitation" hypothesis and the "motor" hypothesis. According to the "motor" hypothesis, high expectancy will result in deeper sensory suppression [Figure 8.1(a)]. Importantly, the "sensory facilitation" hypothesis makes exactly the opposite prediction. High expectancy should result in enhanced perception for the attended right hand [Figure 8.1(b)].

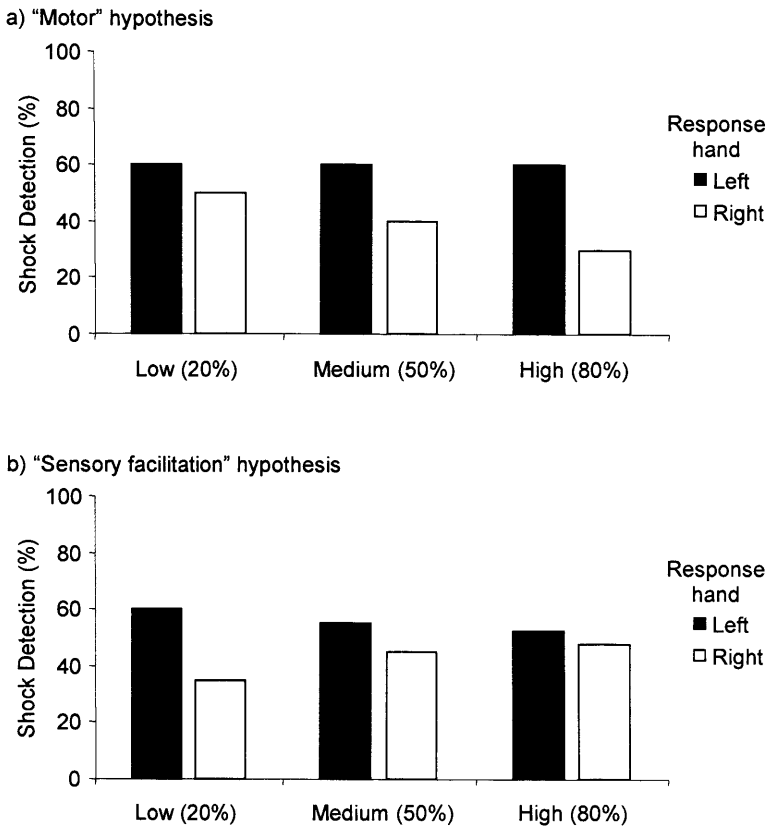

Figure 8.1. Schematic of possible shock detection rates when the probability of a right hand movement is low, medium and high. a) "Motor" hypothesis - Shock detection rates for the right hand decrease with the probability of a right hand movement and conversely b)) "Sensory facilitation" hypothesis - Shock detection rates for the right hand increase with expectancy. Note that the difference in shock detection performance for the left and right hand is much greater in the high expectancy condition for hypothesis (a) than hypothesis (b).

8.3 Materials and Methods

8.3.1 Subjects

Sixteen paid subjects took part with ethical committee approval. Four subjects were excluded because of unstable detection rates (+/-15% of pre-experiment levels). The twelve remaining subjects were included in the final analysis (4 female). Eight subjects were right-handed; the mean age was 21.2 (SD = 3.6) years.

8.3.2 Procedure

The subject's left and right hands were positioned with each index finger resting on a pivoting plate (see Figure 8.2) fixed to a potentiometer. Movement of each finger was mechanically limited to 20° and direct vision of the hands was prevented.

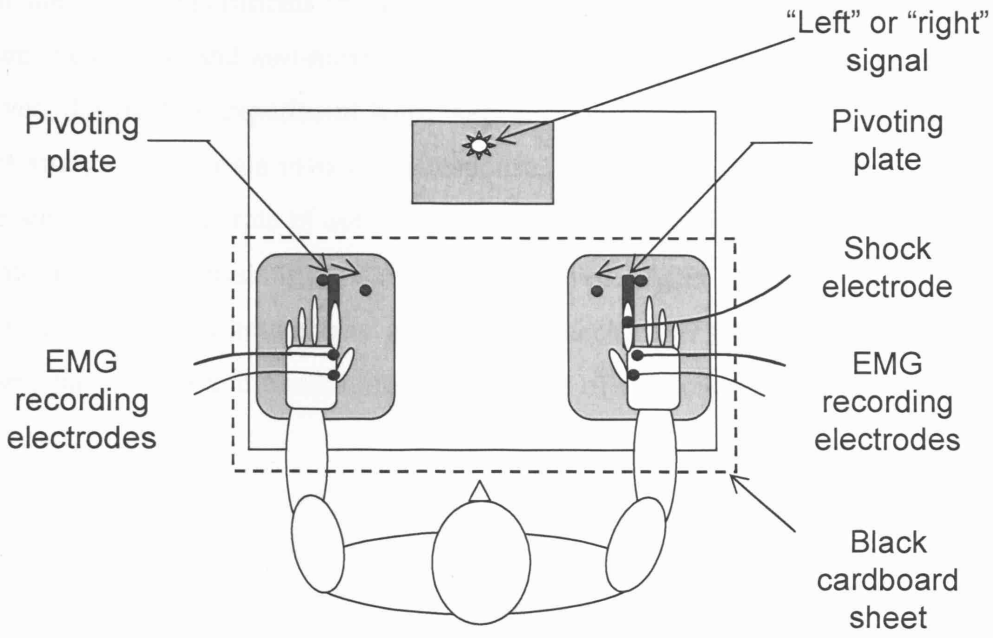

Figure 8.2. Experimental set-up. Subjects placed their two hands on mouse-pads with each index finger resting on a pivoting plate. The Go (move 'right' or 'left') signal was presented via an LED positioned approximately 1 m in front of the subject. A black cardboard sheet (dotted rectangle) was placed over the subject's hands and lower arms to prevent direct vision. Note that shocks were delivered only to the right index finger.

The practice block consisted of 20 right and 20 left movement trials with 4 (no shock) catch trials (2 left and 2 right). The shock stimulus was adjusted during the practice to a level where subjects felt approximately 90% of shocks to the right hand at rest (Williams et al., 1998). There were 3 blocked conditions with a low (20%), medium (50%) or high (80%) probability of a right hand movement. No information was given regarding the probability of a right hand movement. A brief acoustic tone marked the onset of the trial.

After 1000 ms, a red or green Go signal was presented for 700 ms (Figure 8.3). For half of the subjects, a green LED indicated a right hand movement while red indicated a left hand movement. For the remaining half of the subjects this stimulus-response mapping was reversed. The shock was delivered to the right index finger 50 ms before each subject's mean response time from the practice block. On each trial, subjects moved either the right or the left index finger in response to the colour of the Go signal. Note that in the other experiments in this thesis that the difference in shock detection rates between movement and *non*-movement trials formed a measure of sensory suppression. However, during this experiment there were no non-movement trials. Therefore, in the current study, as all trials involved a response, sensory suppression was defined as the difference between the rate of detecting shocks on the right hand on trials when the right hand moved, and on trials when the left hand moved. After each trial, subjects reported verbally ('yes'/'no') whether they perceived a shock. No feedback was given. Trials were self-paced with a minimum intertrial interval of 1 second before the start of the next trial.

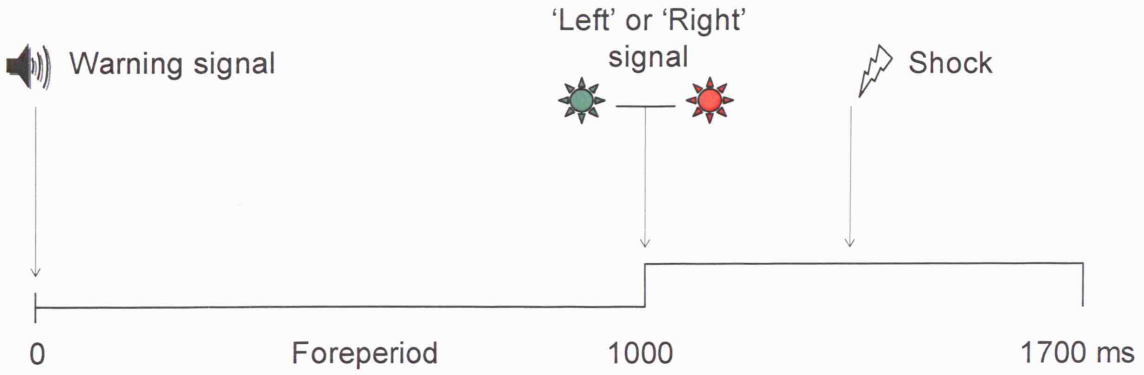

Figure 8.3. Experimental design. An acoustic warning signalled the start of the trial. After a preparation interval of 1000 ms, either a 'left' or 'right' LED signal was presented. Subjects moved their left index finger if the 'left' signal was presented and moved their right finger if the 'right' signal was presented. The probability of a right finger signal on each trial was manipulated between blocks (low, 20%; medium, 50%; or high, 80%). The shock was delivered to the right finger at each subject's mean reaction time minus 50 ms.

Subjects performed a practice block of 44 trials, followed by 6 experimental blocks of 54 trials. The probability of a right hand movement was 20% (low), 50% (medium) or 80% (high) in each of the experimental blocks. Subjects completed two blocks at each of the probability rates. In the low expectancy block there were 40 left movement trials and 10 right movement trials. In the medium expectancy block, there were 25 left and 25 right movements while in the high expectancy block, there were 10 left movement trials and 40 right movement trials. Each block also contained 4 catch (no shock stimulus) trials divided between left and right movements. Subjects were given no information as to the likelihood of a right hand movement. The order of the trials and of the blocks was randomised.

8.4 Results

On catch trials with no shock stimulus, only 1.7% false positive detections were recorded. A 3 x 1 ANOVA showed that there was no difference in the number of false positives on catch trials between conditions $F(2,22)=0.186$; $p=0.835$ (see Table 8.1).

Table 8.1. The number of false positive catch trials pooled across subjects when the probability of a right hand movement was low, medium and high.

Trial Type	
Low (20%)	2
Medium (50%)	1
High (80%)	2

Errors of omission (i.e. movement trials without movement during the response window) occurred on 0.8% of movement trials. These trials were excluded when measuring the effects of sensory suppression.

8.4.1 Stability across time of perceptual performance at rest

The pre-experiment shock intensity threshold (mean pulse-width = 22.3 μs) did not differ from the post-experiment shock intensity threshold (mean pulse-width = 21.8 μs); $t(11)=0.481$; $p=0.640$.

8.4.2 Reaction times

The mean overall reaction times for the low (20%), medium (50%) and high (80%) expectancy conditions of right hand movement were 325 (SD=41), 329 (SD=40) and 330 (SD=42) ms respectively. The mean overall shock to EMG onset intervals were 67 (SD=41), 70 (SD=40) and 71 (SD=42) ms respectively. This difference was not significant $F(2,22)=0.415$; $p=0.618$, indicating accurate delivery of the shock-timing across conditions. The shock was delivered prior to EMG onset on 77.0% of all movement trials and the remaining 23.0% were excluded from the analysis for sensory suppression effects so that only premotor sensory suppression trials linked to preparation processes were analysed. Sensory suppression which occurs after EMG onset could involve peripheral masking and is not of interest here.

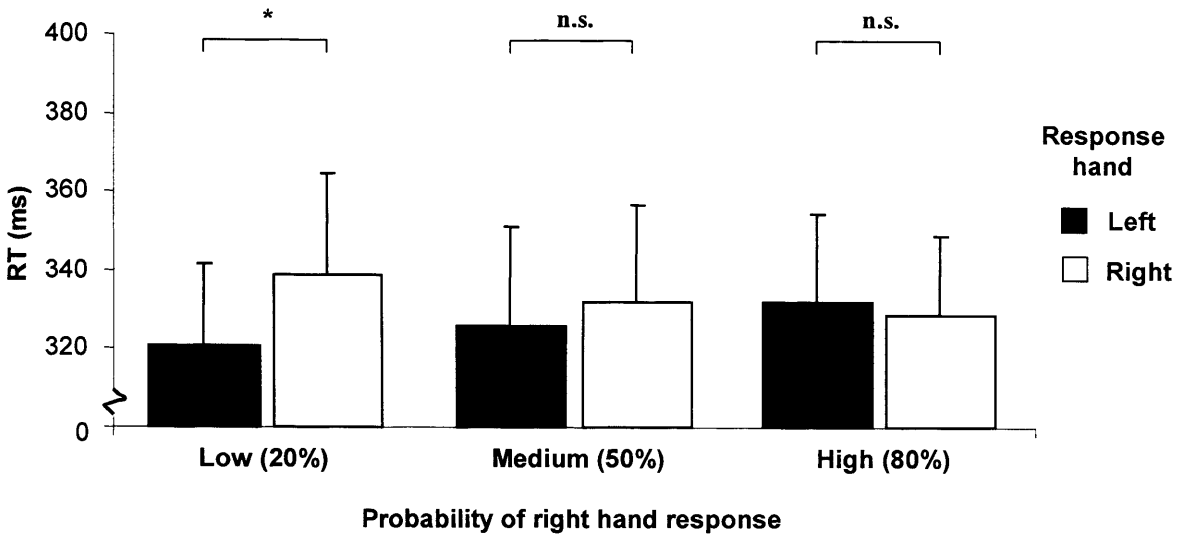

Figure 8.4. Mean reaction times (RT; +/- SD) for the left and right hands when the probability of a right hand movement was low, medium and high.

The reaction times for the left and right hands were explored. The mean overall reaction times for the left and right hands were 326 (34) ms and 333 (37) ms respectively. Figure 8.4 shows reactions times for the left and right hands per condition. Reaction times were subjected to a 2 x 3 repeated measures ANOVA for the factors hand (left vs. right) and expectancy of right hand response (low, medium or high). There was no main effect of hand $F(1,11)=1.479$; $p=0.249$. The factor expectancy of right hand response also did not reach significance $F(2,22)=0.061$; $p=0.899$. However, the interaction hand by expectancy of right hand response was significant $F(2,22)=4.494$; $p=0.027$. Follow-up t tests showed the right hand was significantly slower than the left hand for the low expectancy condition $t(1,11)=2.682$; $p=0.021$. Reaction times were similar for the medium $t(1,11)=0.849$; $p=0.414$ and the high expectancy conditions $t(1,11)=0.395$; $p=0.701$.

8.4.3 Errors

The rate of errors for the low, medium and high expectancy conditions were 3.1%, 1.5% and 2.5% respectively. There was no difference in the number of errors between

conditions F(2,22)=2.491; p=0.125. Sixty-six percent of errors involved subjects moving both hands fully or partially during a trial. The remaining 34% of errors involved responding with the wrong effector.

8.4.4 Shock detection

Figure 8.5 shows shock detection rates for the right hand prior to left and right hand movements per condition. A 2 x 3 repeated measures ANOVA for the factors hand (left vs. right) and expectancy of right hand response (low, medium or high) showed no main effect of hand F(1,11)=1.732; p=0.215 nor expectancy F(2,22)=0.203; p=0.791. However the interaction hand by expectancy was significant F(2,22)=4.798; p=0.033. Follow-up t tests showed that shock detection rates were worse during right hand movements for the low t(11)=1.871; p=0.044 and medium conditions t(11)=2.275; p=0.022 demonstrating the effector-specific nature of sensory suppression. Crucially, in the high expectancy condition, there was no difference in shock detection rates for left and right hand movements t(11)=0.550; p=0.308 (all values one-tailed). The focused effects of sensory suppression were absent when the probability of moving the right hand was high. Inspection of Figure 8.5 suggests that this may have been due to impaired detection of shocks on rare left-hand trials, rather than any change in detection on frequent right-hand trials. However, exploratory t tests found no difference in shock detection rates between the medium and high conditions for the left hand t(11)=1.791; p=0.101 nor for the right hand t(11)=1.536; p=0.153.

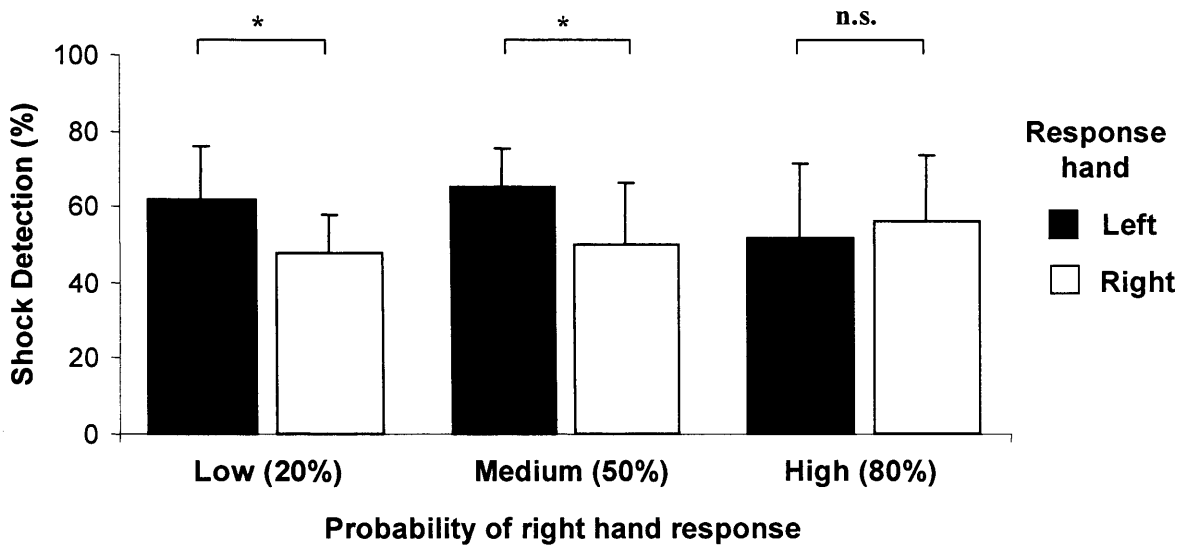

Figure 8.5. The mean percentage of shock stimuli detected (+/- SD) on the right hand prior to left and right hand movements for the low, medium and high (probability of a right hand movement) conditions.

8.4.5 Time-dependent changes in the detection of stimuli applied to the moving digit

Figure 8.6 shows that the effects of right index finger movement on the ability of subjects to detect the weak shock stimulus to the moving finger were not uniform over time. For the right hand, performance declined in accordance with premovement sensory suppression beginning approximately 100 ms before EMG onset with the peak decrease in perceptual performance coinciding approximately with movement onset i.e. 50 ms after EMG onset. Furthermore, linear regression equations with slopes significantly different from zero ($p=0.002$; $p=0.018$ and $p=0.009$ for the low, medium and high expectancy conditions respectively), indicated that for the right hand there was a time-dependent change in performance for all conditions. A different pattern of perceptual modulation was observed when the left finger was abducted. The slopes for the left hand were not significantly different from zero ($p=0.280$; $p=0.224$ and $p=0.501$ for the low, medium and high expectancy conditions respectively), indicating no time-dependent change in performance. This finding replicates Experiment 2 and the original work by Williams et al. (1998), demonstrating that while there is a relatively small and significant decrease in perceptual performance for a more distant body part, it does not seem to be time-

dependent. Note in Figure 8.6(a) and (b) that shock detection performance for the right hand is generally worse than the left hand. This difference in performance demonstrates that sensory suppression effects are focused on the moving hand. However, when the probability of a right hand movement is high ([Figure 8.6(c)], the difference between the two hands is not so clear. There is a considerable overlap in performance for both hands suggesting that effector-specific sensory suppression effects are switched off.

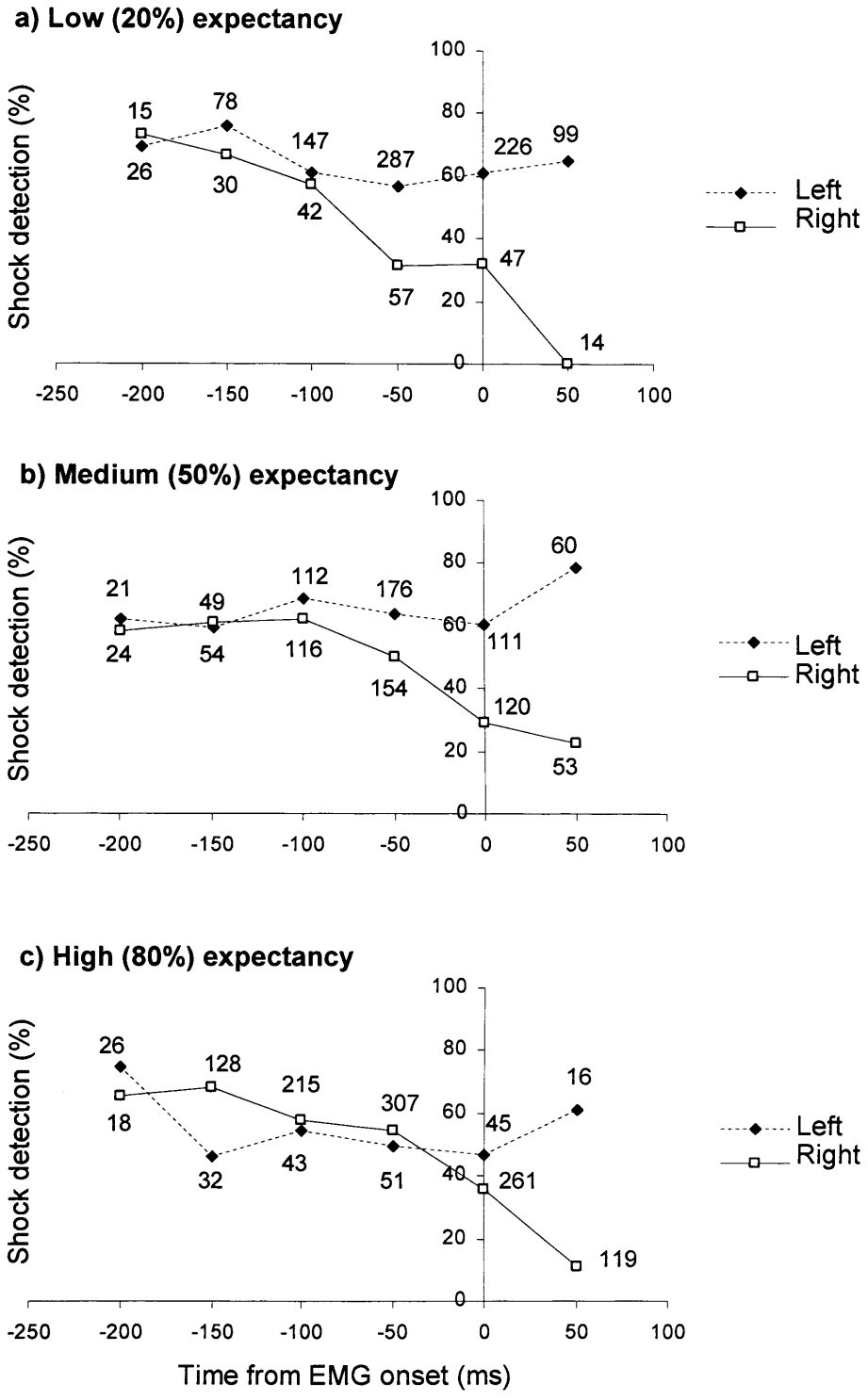

Figure 8.6. Effects of left and right hand movement on the detection of fixed intensity stimuli applied to the moving right index finger when the probability of a right hand movement was a) low, b) medium and c) high. Detection performance over time is plotted relative to the onset of EMG (0 ms); a negative number indicates that the shock precedes EMG onset. The values next to the data points refer to the number of trials represented at that data point.

8.5 Discussion

The aim of the present study was to investigate sensory processing under conditions where the motor system was required to switch movement from one effector to the other under varying degrees of expectancy. Manipulating expectancy required a change in the planned effector i.e. inhibition of the planned movement and facilitation of the alternative movement. Two competing hypotheses were proposed; a "motor" hypothesis and a "sensory facilitation" hypothesis (see Figure 8.1). The "motor" hypothesis predicted a *decrease* in shock detection performance as the probability of a right hand increased (see Chapters 3 and 4). Conversely, the "sensory facilitation" hypothesis predicted an *improvement* in shock detection performance as the probability of a right hand increased due to attentional enhancement, in accordance with the premotor theory of attention (Rizzolatti et al., 1994). The results show that there was no decrease in shock detection rates with increasing probability of movement (Figure 8.5); therefore a key prediction of the "motor" hypothesis was not borne out. In fact, there was a non-significant trend for shock detection performance to improve with increased probability of movement of the right (shocked) hand. These results are more consistent with the "sensory facilitation" hypothesis ([Figure 8.1(b)]. However, great caution is necessary when drawing a conclusion from this experiment. Overall, the results are not clear and seem to suggest a pattern of behaviour somewhere between the two competing hypotheses.

Consistent with Experiment 2 and Williams et al. (1998), shock detection rates on the right (stimulus) hand were lower than on the left (non-stimulus) hand in the low and medium expectancy conditions, demonstrating the effector-specific nature of movement-related sensory suppression. However, when the probability of a right hand movement was high, there was no difference in shock detection rates for the left and right hands and the effector-specific characteristics of movement-related sensory suppression were absent. These results are more consistent with the "sensory facilitation" hypothesis ([Figure 8.1(b)].

The present study supports the finding that reaction time becomes faster as a result of a high expectancy in comparison to medium and low expectancies (see Figure 8.4) consistent with other reports (e.g. Larish and Stelmach, 1982; Leis et al., 2005). Consistent with many real-world situations, subjects were given no information about the varying probability of responses in each block. Nevertheless, the reaction time value of the medium condition that contained no expectancy information was between the values of the high and low expectancies. This finding suggests that movement preparation enjoyed a benefit in the high and a cost in the low compared to the medium expectancy condition. Our subjects presumably took advantage of the probability of a right hand movement in advance of the Go signal in order to plan and prepare for that movement. That is, subjects prepare in advance for what they think, on the basis of experience, is coming next (Cleeremans et al., 1998). They can predict or anticipate the next trial based on some estimation of the probability of the response required on the next trial.

It could be suggested that the modulating effects of movement probability on sensory detection performance could be explained by differences in RTs across conditions. Movement-related gating is also a function of the kinematics of the movement, with faster movements producing larger gating effects (Angel and Malenka, 1982). While the high expectancy condition produced faster reaction times for that hand, our data showed no evidence of improved somatosensory detection for the right hand in the high expectancy condition. Indeed, we found a trend in the opposite direction (Figure 8.5). Plotting the results in a response-locked fashion (Figure 8.6) confirmed the basic result. Therefore, an explanation of our results in terms of reaction time artefacts can be ruled out.

According to the premotor theory of attention, the control of goal-directed movements and the control of attention are closely linked, because they are implemented by common structures, with different control mechanisms specialised for different types of movements, and for different parts of space (Rizzolatti et al., 1994). Central to the premotor theory is the claim that shifts of attention are triggered whenever these shared control structures are activated during response preparation. Evidence in favour of the premotor theory comes from saccadic eye movement studies demonstrating that

attentional shifts towards saccade target locations are triggered during saccade preparation even before the eyes have begun to move (Hoffman and Subramaniam, 1995; Irwin and Gordon, 1998), resulting in superior performance to visual (Deubel and Schneider, 1996), auditory (Rorden and Driver, 1999), or tactile stimuli (Rorden et al., 2002) at this location. The premotor theory also explicitly claims that shifts of attention are linked to manual response programming. (cf., Berti and Frassinetti, 2000; Butler et al., 2004; Halligan and Marshall, 1991). In the present experiment, the spatial aspects of attention are minimal. Both left and right signals are presented centrally, so subjects do not really shift attention *to* the left or right. However, by varying the probability of a right hand movement across blocks, the present experiment clearly manipulates response programming, or 'intention'. To this extent, the data are in agreement with the premotor theory of attention and point to modulation of sensation by motor expectancy.

Computational forward models of sensory attenuation (e.g. Wolpert, 1997) can mimic sensory feedback and the outcome of an action can be estimated and used before actual sensory feedback becomes available. This prediction can be used to anticipate and compensate for the sensory effects of movement; attenuating the component that is due to self-movement from that due to changes in the outside world. In the high expectancy condition when the probability of a right hand movement is high, forward models would predict a right hand movement, and might therefore attenuate sensory inputs from the right hand. Interestingly, we found similar attenuation of sensory inputs from the right hand when subjects expected to move the right hand but in fact moved the left in response to rare left signals. On such trials, the sensory attenuation was more consistent with the motor expectation generated across the entire block, and less inconsistent with the specific motor command sent in response to the signal.

In conclusion, attention and motor preparation are two intimately linked processes though they can be dissociated (Boussaoud 2003; Simon et al., 2002). Our results suggest that attention can override the focused nature of sensory suppression under certain conditions. Typically effects of expectancy have been studied using reaction times. Here we show that sensory processing is also affected.

Here, we investigated the sensory consequences of expecting, and possibly then reprogramming, a particular response. However, certain situations place other demands on movement preparation. Behaviour often involves stringing a series of actions together to form a sequence. In the next chapter, we explore the effects of action complexity on premovement sensory suppression.

Chapter 9: Sensory suppression and the preparation of motor sequences

9.1 Abstract

This chapter investigates whether sensory suppression is organised for individual movements or for entire action sequences composed of several movement elements. The latter result would suggest a hierarchical cognitive component of sensory suppression. In this *prior instruction* task, subjects were informed at the start of the trial if they should move or not. Subjects had to detect weak shocks which were delivered after the signal on some trials. On movement trials, subjects performed blocks of sequences of right and left finger movements. We investigated if any sensory suppression related to the second, third or fourth element in a sequence can be found before the first element is executed. There was no difference in shock detection rates for two critical conditions. Thus, sensory attenuation was only affected by the first part of the movement sequence. The remaining three elements in the sequence had no effect on sensory attenuation. This result suggests that sensory suppression is programmed in advance of the immediate impending movement only. Sensory suppression appears to arise after the level of the cognitive-motor representation that underlies sequencing.

9.2 Introduction

Everyday activities can recruit a variety of sensory and cognitive processes that are involved in assembling a series of movements into an action. Typing is an example of elements organised serially at different levels. Individual letters are typed serially to form a syllable, these syllables are chained together to make a word, words are combined to form sentences. In other forms of motor action, simple movements are produced serially in order to execute a more complex integrated set of movements e.g. combining a number of single key strokes on the piano to form a pleasing melody. An impressive aspect of human cognition is that such arbitrary sequences can be learned and executed with ease. Lower-order units of actions such as simple movements can be ordered serially within higher units to produce more integrated and complex motor sequences.

The simplest involuntary movement is the reflex. The very existence of the reflex suggested to Sherrington (1906) that our complete behaviour might be built from reflex chains with the response to one reflex triggered by the next and so on. Thus, chains of reflexes could occur as a result of feedback to the central nervous system. This is a clear example of the 'bottom-up' control of behaviour. Although the response-chaining

hypothesis does not seem to hold any longer, it gave rise to an increase in interest in the top-down organisation or "programming" of complex behaviour (Brunia and van Boxtel, 2000).

The very existence of anticipatory control of entire sequences is a strong argument against the chaining theory and in favour of the existence of motor programmes (Rosenbaum, 1980). In grasping a ball, the fingers and the hand are shaped in advance in order to be able to catch and hold the ball in the closing hand (Jeannerod, 1997). Thus, while the hand is directed towards a certain point in space, the next step is already prepared. These anticipatory effects in the behavioural output are considered a strong argument in favour of the existence of motor programmes (Brunia and van Boxtel, 2000). Keele (1968) defined a motor programme as "a set of muscle commands that are structured before a movement sequence begins, and that allows the entire sequence to be carried out uninfluenced by peripheral feedback". These motor programmes are thought to be ordained in a hierarchical way (Rosenbaum et al., 1983). The hierarchy defines the identity and order of the commands in the musculature, represented as terminal nodes in the hierarchy. Execution of a programme is either based upon a linear readout of the terminal nodes, or the terminal nodes have to be passed again and again, so that all members of the hierarchy are activated repeatedly. The specification of response complexity must be implemented before the movement begins. Therefore, the reaction time to the first of a series of responses increases with the length of the series even though the first movement remains identical. Sternberg et al. (1978) presented subjects with a short list of digits or words sequentially at a rate of about one item per second. They found that the mean latency to begin to repeat the list out loud increased linearly with the number of words in the list at a rate of 12 ms per word (the so-called 'length effect'). Thus the time to start saying a five word list was about 50 ms greater than the time to begin uttering a single word. Sternberg et al. (1978) proposed that a representation of the entire response appropriate for controlling its execution (a "program") is constructed before the start of each response. This programme consists of a set of "linked subprograms", one for each unit of the response and is stored in a special motor buffer. According to this hypothesis, the construction of the programme as a whole is

accomplished prior to the Go signal. However, the sub-programme for the first unit can only be retrieved in the programme once the Go signal has occurred.

Various models of motor programming explain the RT length effect in different ways. One model attributes response complexity effects to differences in the amount of time required to *transfer* the motor programme from long-term memory (LTM) into a short-term memory (STM) motor buffer (Klapp, 1976; 1978). A second model explains response-complexity effects in terms of the time needed to *edit* the programme while it resides in the buffer and to traverse its hierarchical structure from the highest to the lowest node that controls the first element of the movement (Rosenbaum et al., 1986). Finally, according to a third model (Sternberg et al., 1978); the processes affected by response complexity involve *searching* the motor programme buffer for the subroutine that controls the first element of the movement and then performing a second set of "unpacking" operations on the subroutine. All these models have in common the idea that a single representation for an entire sequence is first stored in some kind of motor memory *buffer*. Just prior to movement, the contents of the buffer are processed or *unpacked* in serial fashion and dispatched to the motor circuits (Sternberg et al., 1978).

9.2.1 Prefrontal motor buffer

In the last decade, neurophysiological evidence for the existence of a buffer has been advanced. For example, Averbeck et al. (2002) provided evidence that all elements of a movement sequence are represented in an orderly fashion in the prefrontal cortex (PFC) before the action begins. The activity of individual neurons was recorded simultaneously in the PFC while monkeys copied geometrical shapes (e.g. a square or a triangle) shown on a screen. The monkeys drew the shapes as sequences of movement segments. Each of these segments was associated with a distinct pattern of neuronal ensemble activity. These patterns were present during the time preceding the actual drawing suggesting that the sequence is prepared and loaded before the action actually begins. These results provide strong evidence that all elements in a sequence are simultaneously represented in

the form of parallel response activation in a prefrontal buffer before movement begins. In other words, "the elements of a sequence are activated before the order is imposed on them" (Lashley, 1951).

9.2.2 Unpacking the buffer

Neurophysiological evidence has also been found for the 'unpacking' of the prefrontal buffer, explaining the transition from composite but non-ordered storage of a representation to a specific ordering of action. Shima and Tanji (1998) trained monkeys to perform three different arm movements. The movements were individually initiated after a variable time interval. Inactivation of either the supplementary motor area (SMA) or the pre-SMA by muscimol injection greatly impaired the monkeys' ability to perform the three movements in the correct order. In contrast, the monkeys retained the ability to perform the three movements without difficulty when individually guided by a visual cue. However, the temporal arrangement of the three movements in sequence was very impaired. Single cell recordings revealed three types of neuronal activity of particular interest (Shima and Tanji, 2000). First, activity changes were found that were selective for a particular sequence of the three movements that the monkeys were prepared to perform. This ordered activity ceased as soon as the monkeys initiated the first movement. These sequence-selective neurons suggest that particular ordered patterns of the elements are represented as a single unit for the purposes of programming action. Second, they found interval-selective activity that appeared in the transition between one particular movement and the next. Finally, they found neuronal activity representing the rank order of the three movements arranged chronologically i.e. the activity differed selectively during and before initiation of the first, second or third movements on individual trials (see Figure 9.1).

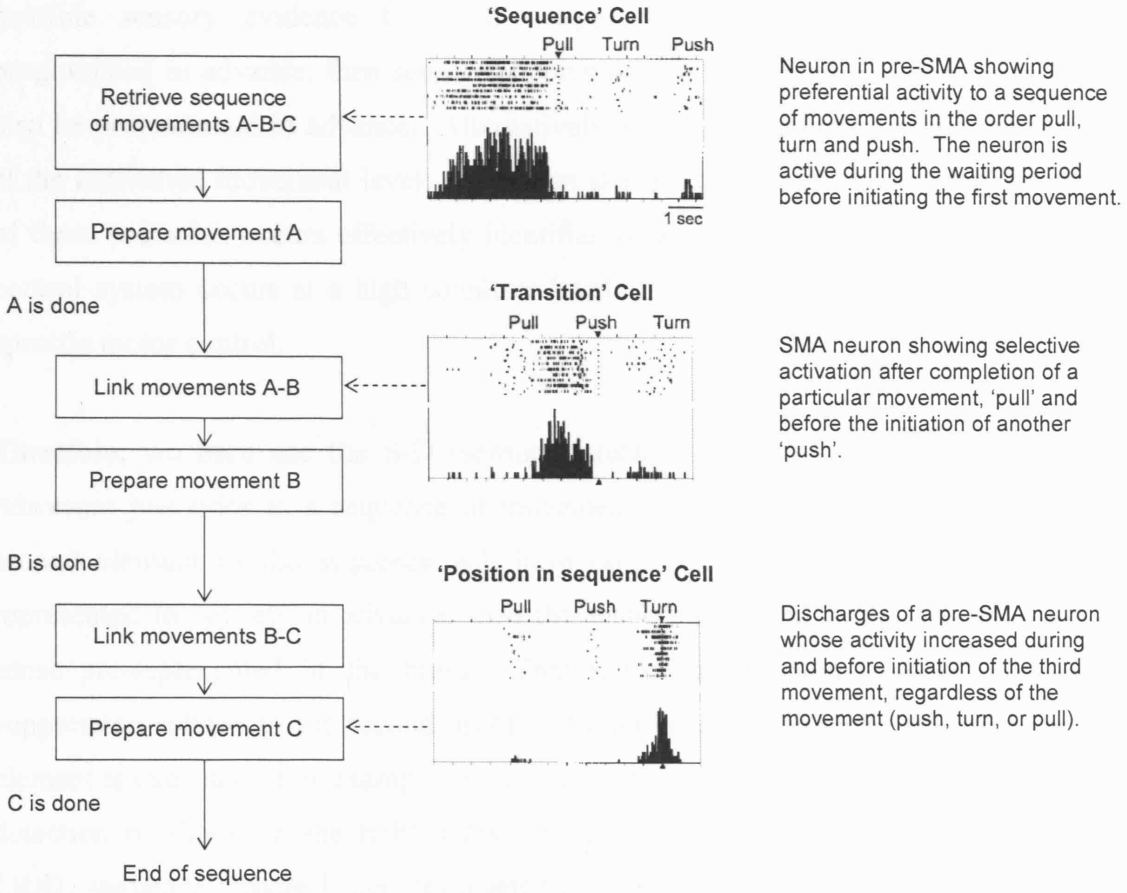

Figure 9.1. Schematic showing how information stored in a memory buffer might be 'unpacked' and serially organised in readiness for the performance of three movements in a sequence with the order A, B, and C. The information for the correct sequence should lead to a chain of events giving rise to the orderly delivery of the three movements in the correct order. Each movement is externally triggered by a signal. The raster displays represent discharges for individual neurons (from Shima and Tanji, 2000).

Action need not occur immediately. The stored representation of an entire sequence can be held in the PFC-buffer while waiting to move (Averbeck et al., 2002; Fuster, 1997). Actualising the sequence in the correct order signifies the implementation and the execution of one or more motor programmes, for which the SMA, the PMC, and M1 have to be activated (Shima and Tanji, 2000; Rosenbaum, 1985).

Behavioural evidence for advance preparation of motor sequences comes largely from sequence length effects in RT (e.g. Sternberg et al., 1978). This chapter investigates the

possible sensory evidence for such pre-programming. If entire sequences are programmed in advance, then sensory suppression for all the movement elements might also be programmed in advance. Alternatively, sensory suppression might be organised at the individual movement level, rather than at the sequence level. Investigating which of these scenarios occurs effectively identifies whether sensory prediction in the motor control system occurs at a high cognitive level of entire action or at the lower level of specific motor control.

Therefore, we here use the S-D (sensory-detection) task to investigate the cognitive processes just prior to a sequence of movements. Of central interest is the effect the second element of the sequence will have on shock detection rates. If chunks are represented in entirety in advance, then the second, third and fourth elements are in a sense pre-represented in the brain. That raises the question whether any sensory suppression related to the second, third or fourth element can be found before the first element is executed. For example, as sensory suppression is specific to the moving digit, detection of shock on the right index (stimulus) finger would be worse just before a LRRL sequence (where L denotes a left finger movement and R denotes a right finger movement) than before an LLRR sequence. That result would suggest that sensory suppression is programmed in advance for whole sequences, not just the immediate impending movement. We plan to compare shock detection rates delivered prior to movement for these two critical conditions (Figure 9.2).

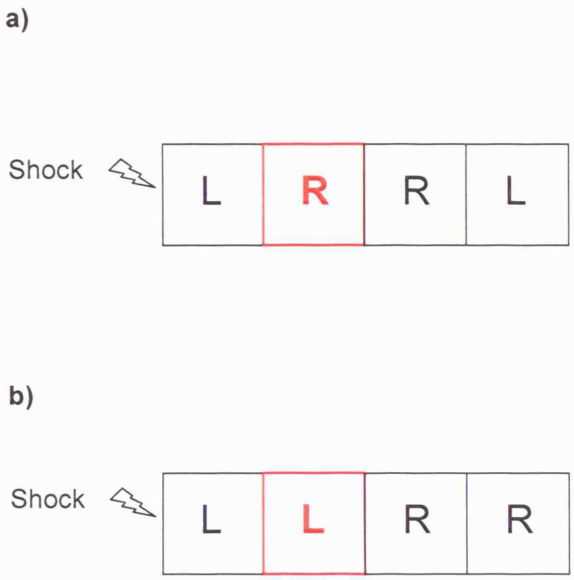

Figure 9.2. Schematic showing the two critical conditions. In both sequences, the identity of the first movement and the overall contents of the sequence are the same. Note that the second element of the sequence, shown here in red, is a) right (R) and b) left (L). The shock is delivered to the right index finger.

9.3 Materials and Methods

9.3.1 Subjects

Eleven paid subjects took part. Subjects gave their written consent, and local ethical guidelines were followed. The data from 2 subjects were excluded because their detection of cutaneous shocks at rest was unstable across the experiment (post-test detection varied more than +/-15% from pre-experiment levels). Data from the remaining 9 subjects (6 female, all right-handed, mean age 21.8 (SD = 3.1) years) were included in the final analysis.

9.3.2 Procedure

The set-up was the same as for Experiment 7 (Chapter 8). Briefly, the subject's right and left hands were positioned on 2 mouse pads with each index finger resting on a small pivoting plate (1.8 x 10 cm; see Chapter 8; Figure 8.2). There were 5 blocked experimental conditions; namely L (where L denotes a single over and back movement of the left index finger), LRRL (i.e. a movement sequence consisting of a left index finger movement, followed by a double movement of the right index finger and finishing with a left finger movement), LLRR (left, left, right, right), R (right only) and RLLR (right, left, left, right). The two critical conditions were LRRL and LLRR (Figure 9.2). During a sequence trial, subjects were required to make the 20 degree movement for each of the four elements in a sequence.

A verbal instruction "move" or "don't move" was given by the experimenter at the start of each trial (see Figure 9.3). After a delay of 1000 ms, the Go signal (green LED for 2000 ms) was preceded at 290 ms intervals by 2 brief (10 ms) countdown flashes (ready-steady-go). These were included to minimise the subject's time uncertainty about when to respond and to allow maximum preparedness for action (Sternberg et al., 1978). The Go signal remained on throughout the 2000 ms response window. The shock stimulus was delivered 50 ms before each subject's individual mean reaction time which was calculated separately for each trial type during the practice block. Subjects were instructed to perform the required movement or sequence of movements of the index finger as soon as possible after the onset of the 'Go' signal on movement trials and to make no movement on non-movement trials.

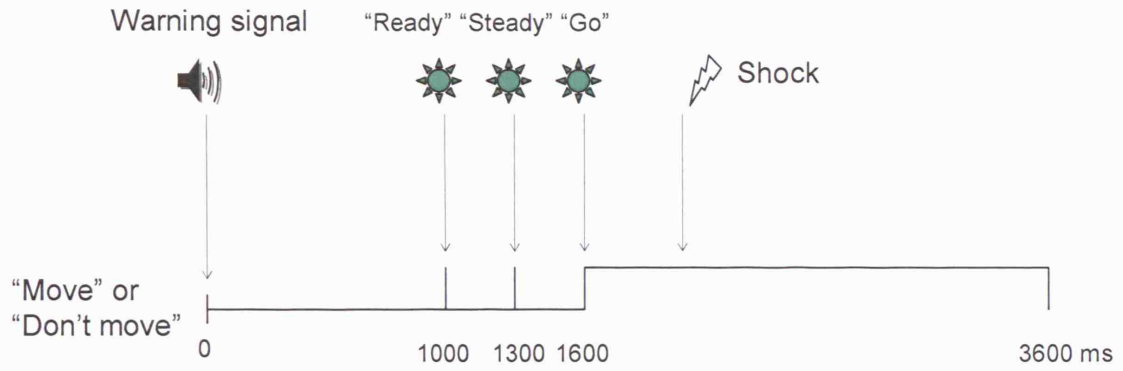

Figure 9.3. Experimental design for each trial. After a delay period of 1000 ms, a countdown LED was flashed twice ('ready', 'steady') briefly. This was followed by the Go signal for 2000 ms. The shock stimulus was delivered 50 ms before each subject's mean RT.

At the end of each trial, subjects reported verbally by responding 'yes' if they perceived a stimulus or 'no' if they did not. Their response was entered into the computer by the experimenter and stored with each trial. No feedback was given. The experimenter initiated each trial ensuring an intertrial interval of at least 1 sec. After completing an initial practice block of 44 trials, subjects completed 5 experimental blocks of 44 trials, one block for each condition. Occasionally, it was necessary to fine-tune the intensity level of the stimulus during the practice block but this was then held constant through the experimental blocks. At the start of each sequence block, subjects practiced the upcoming sequence for approximately 20 practice trials and then during the experimental blocks, performed the sequence from memory. Each block consisted of 30 movement trials and 10 non-movement trials. A further 4 catch trials (no stimulus) were divided equally between Go and NoGo trials. The order of the blocks and the order of the trials were randomised for each subject.

9.4 Results

A typical LRRL sequence trial is shown in Figure 9.4. The reaction time was estimated from the onset of EMG activity for the first movement.

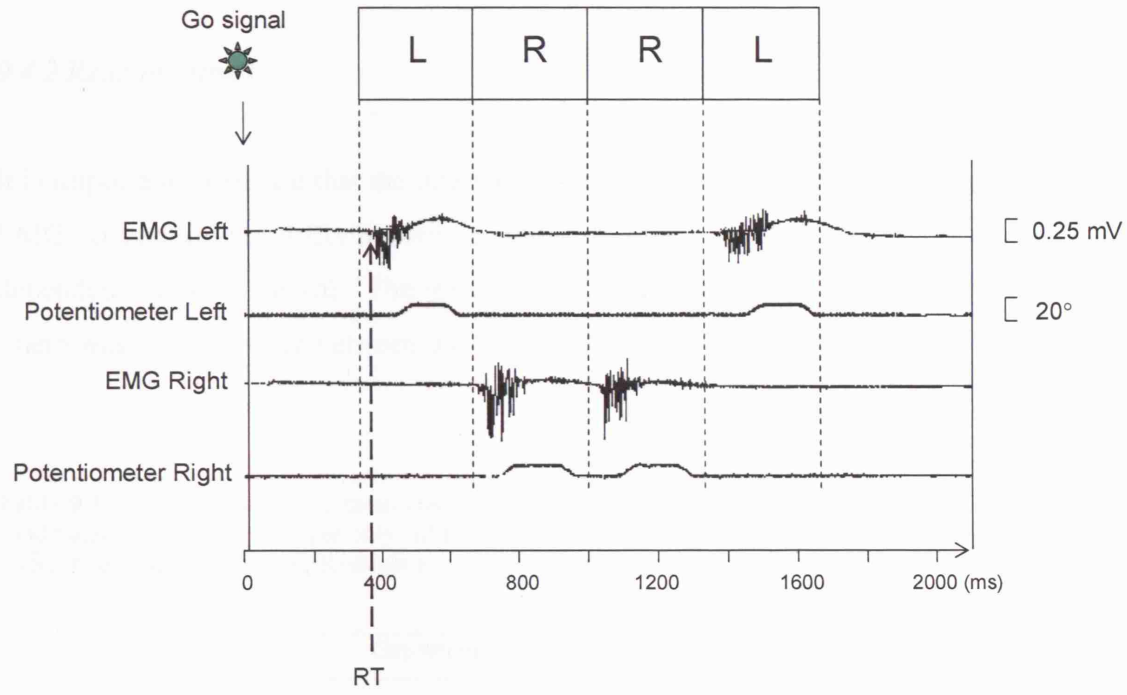

Figure 9.4. Illustrative trial from a single typical subject reacting during a LRRL (left, right, right, left sequence of movements) trial in response to the Go signal showing the EMG signals and potentiometer outputs for the left and right hands. The reaction time (RT) was measured from the onset of EMG activity for the first movement.

On the catch trials no false positives were recorded, indicating that subjects did not guess throughout the experiment. The errors of commission collapsed across conditions (i.e. when a non-movement trial was accompanied by EMG activity) rate was 2.8%. The errors of omission (i.e. movement trials where there was either no movement or where the subject's response occurred outside the 2000 ms response window) accounted for 0.6% of all movement trials.

9.4.1 Stability across time of perceptual performance at rest

The pre-experiment shock intensity threshold (mean pulse-width=21.8 μs) did not differ from the mean post-experiment threshold (mean pulse-width=22.2 μs), $t(8)=0.839$; $p=0.426$.

9.4.2 Reaction times

It is important to ensure that the interval between the timing of the shock and the onset of EMG activity did not differ between conditions as the magnitude of sensory attenuation is dependent on this interval. The mean RTs for the five conditions are given in Table 1. There was no difference between the RTs for the five conditions $F(4,32)=0.614$; $p=0.609$.

Table 9.1. Table showing the mean reaction time (RT) and standard deviation (SD) for each of the five conditions. L=left index finger only; LLRR = left, left, right, right index finger sequence of movements; LRRL = left, right, right, left; R=Right and RLLR= right, left, left, right.

Condition	Mean RT (SD) ms
L	237 (111)
LLRR	233 (143)
LRRL	220 (123)
R	248 (125)
RLLR	226 (141)

9.4.3 Shock detection

Figure 9.5 shows the percentage of shocks detected for both movement and non-movement trials. Note that shock detection performance before movement is generally better when the first movement is a left hand movement (3 black bars on left) relative to a

right movement (2 black bars on right). This is because sensory suppression is specific to the moving right hand. The percentage of shocks detected was subjected to a 2 x 5 repeated measures ANOVA for the factors movement (move vs. don't move) and sequence (L, LLRR, LRRL, R and RLLR). There was a significant main effect of movement $F(1,8)=26.041$; $p=0.001$. The main effect of sequence approached but did not reach significance $F(4,32)=2.856$; $p=0.060$. Importantly, the movement by sequence interaction was also not significant $F(4,32)=1.722$; $p=0.200$. The planned comparison, as per the experimental hypothesis, revealed no significant difference in shock detection rates between the two critical conditions LLRR and LRRL; $t(8)=0.479$; $p=0.644$.

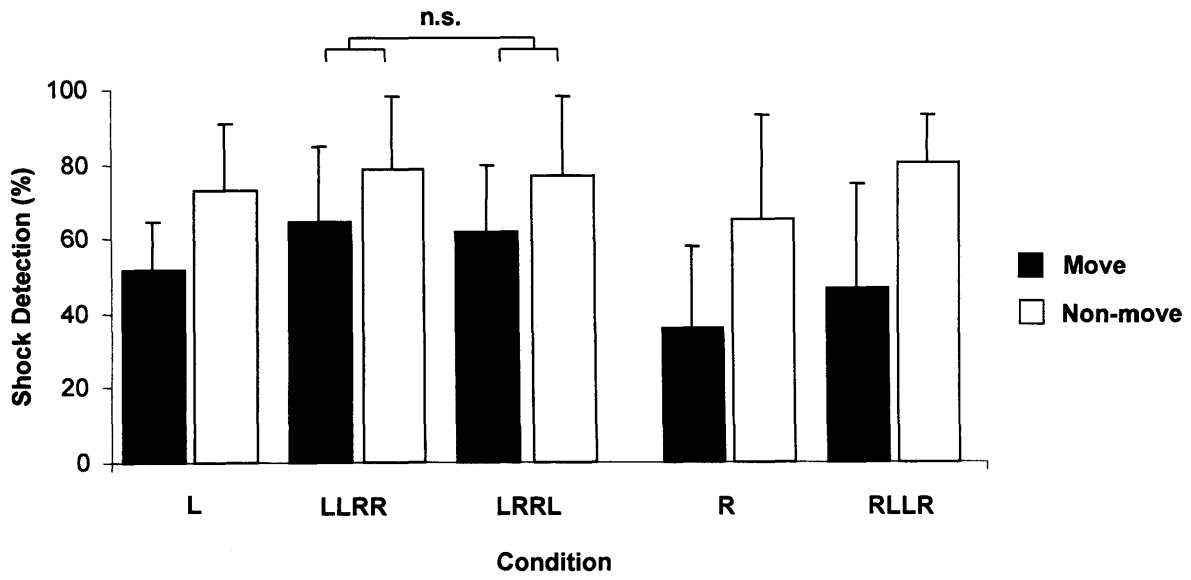

Figure 9.5. Graph showing the mean percentage of shock stimuli (+/- SD) detected for movement (black bars) and non-movement (white bars) trials for the 5 conditions. The planned comparison between the two critical conditions, LLRR and LRRL, was not significant. L=left index finger only; LLRR = left, left, right, right index finger sequence of movements; LRRL = left, right, right, left; R=Right and RLLR= right, left, left, right.

9.4.4 Time-dependent changes in the detection of stimuli applied to the moving digit

Note in Figure 9.6(a) that there is no hint of a downward slope for the LRRL condition. When left finger movement was first, slopes did not significantly differ from zero (L,

p=0.311; LLRR, p=0.788 and LRRL p=0.797) indicating that there was no time-dependent change in performance for left hand movements. In contrast, linear regression equations indicated time-dependent changes in performance with slopes significantly different from zero when right finger movement was first; R (p=0.011) and RLLR (p=0.009). The absence of time-dependent performance in the critical condition (LRRL sequence) is further evidence that sensory suppression is influenced by the impending movement in a sequence only. An impending left movement does not produce sensory suppression in the moving right hand, even when the initial left movement is followed by a right movement. There is no evidence of sensory suppression prior to a sequence in which the target movement occupies second position.

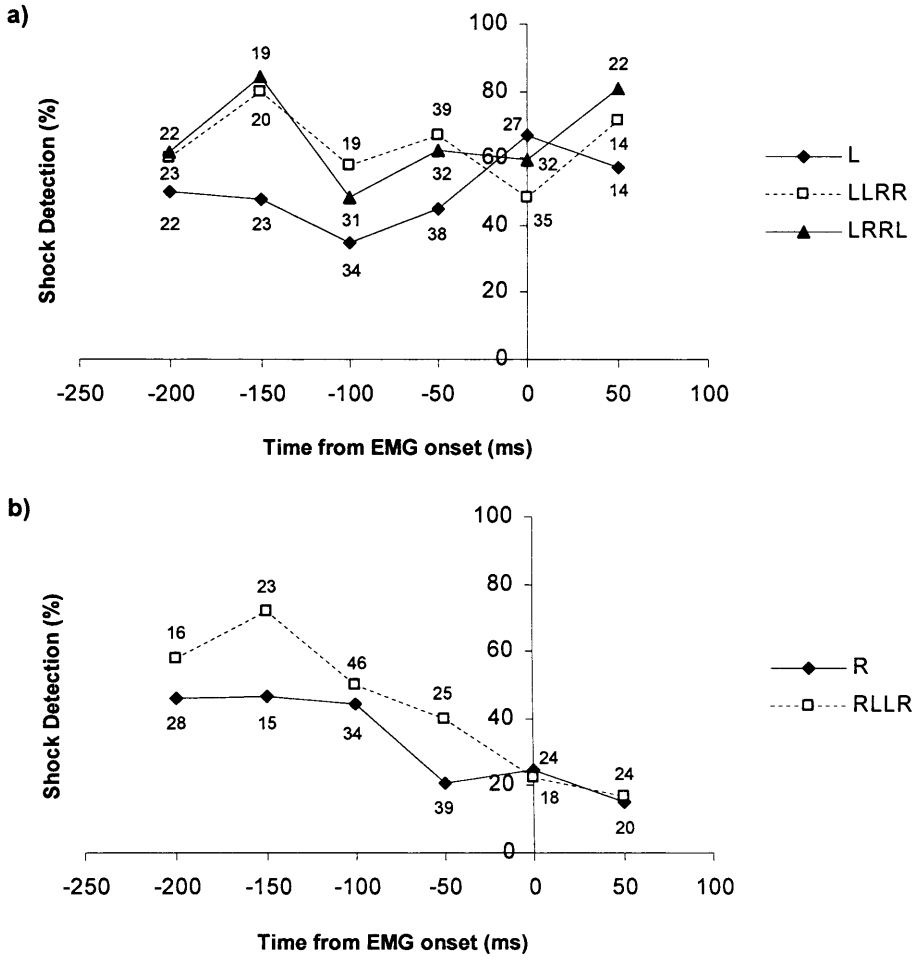

Figure 9.6. Effects of index finger movement on the detection of fixed intensity stimuli applied to the moving and non-moving finger when the first movement was a) a left hand movement and b) a right hand movement. Detection performance over time is plotted relative to the onset of EMG (0 ms). (b) A negative number indicates that the shock precedes EMG onset. The values next to the data points refer to the number of trials represented at that data point, pooled over subjects. L=left index finger only; LLRR = left, left, right, right index finger sequence of movements; LRRL = left, right, right, left; R=Right and RLLR= right, left, left, right.

There was an unexpected trend for shock detection rates to be greater when the shock was delivered prior to a sequence than a single movement [see Figure 9.6(a) and (b)]. For example, shock detection rates for the sequence of movements LLRR or LRRL, tended to be higher than for the single movement "L". The same tendency was present for the right hand. It is not clear why this effect occurred and it should be the topic of future research.

9.5 Discussion

A weak shock delivered just prior to the onset of movement is less likely to be detected than when no movement occurred. This replicates sensory suppression effects previously reported in this thesis (e.g. Experiments 1, 2 and 3; also Williams et al., 1998). The question was raised whether any sensory suppression related to the second, third or fourth element would be found before the first element is executed. Specifically, we asked if the detection of a shock on the right index finger would be worse prior to a sequence in which the target movement occupies the second position (LRRL vs. LLRR sequence). There was no difference in shock detection rates for the two critical conditions. Sensory attenuation was only affected by the first element of the movement sequence. The remaining three elements in the sequence had no effect on the amount of sensory attenuation observed. This result suggests that sensory suppression is programmed in advance of the immediately impending movement only.

An unexpected finding in this study was the absence of the RT length effect with increased complexity of the task. Sternberg et al. (1978) demonstrated that the number of words in a brief utterance can increase the time taken to initiate the utterance even when the speaker knows what to say well in advance of the reaction signal. Here, we found that the mean latency required to perform a four step sequence of movements did not differ from that required to perform a single movement. In fact, there was a small numerical effect in the opposite direction i.e. sequences tended to be performed slightly faster, by approximately 20 ms, than single movements. According to motor programming models, lengthening of the reaction time to the first element in a sequence should take longer than to a single movement as a result of differences in the amount of time required to transfer the motor programme from LTM into a STM motor buffer (Klapp, 1976; 1978), differences in the time needed to edit the programme while it resides in the buffer (Rosenbaum et al., 1986) or due to differences in search times within the motor programme buffer for the subroutine that controls the first element of the movement (Sternberg et al., 1978).

It is not clear why we did not obtain the expected lengthening in RT. However, some tasks do not result in RT increases. If a task is too easily acquired, there is no need to plan and the result can be the absence of a length effect. For instance, Klapp (1996; 2003) found that when the required response (number of syllables or duration of a keypress) was known in advance, increasing the complexity of the action had no effect on RT. Our study utilised practiced, predictable sequences presented in a blocked design in order to achieve automaticity. This procedure may have placed lower demands on preparation processes. Furthermore, previous studies have used speech and typewriting (Sternberg et al., 1978). Speech and typewriting are overlearned skills that involve a very rapid sequencing of a large number of different movement elements. For example, the professional typists who took part in the Sternberg et al. (1978) study, demonstrated test rates in prose typing of about 90 words per minute (7.5 strokes per second). In contrast, it can be argued that the sequences of finger abductions used in the current study were not as overlearned or ballistic, and had relatively slow production rates (approximately three finger abductions per second; see Figure 9.4). Another possibility is that the marginally slower RT for a single movement relative to a sequence of movements may be due to an overall slower movement time in that condition. This cannot be ruled out as the current design did not include checks to verify that there were no differences in kinematic parameters between the different conditions. However, EMG profiles and potentiometer outputs for each subject were visually inspected and no obvious differences were evident. Also, it has been shown elsewhere that providing prior information about a forthcoming movement can strongly affect reaction time, while effects on movement time are negligible (Riehle and Requin, 1989).

The above factors may have contributed to the lack of RT length effect observed here. Interestingly, there was some sensory evidence that subjects were nevertheless engaged in the production of sequences on sequence trials. There was an unexpected tendency for higher shock detection rates just prior to sequence trials compared to singleton trials [Figure 9.6(a) and (b)]. Future research could investigate if there is a linear relationship between the number of items stored in the motor memory buffer and the level of shock

detection performance prior to EMG onset. This would require measuring shock detection performance prior to sequences of several different lengths.

In this experiment, subjects had advance knowledge of the sequence at the start of each trial. This advance knowledge could be manifested in prefrontal cortex as an anticipatory representation of the elements and order of the sequence (Averbeck et al., 2002; Furston, 1997). Once the Go signal is presented, the sequence is then 'rattled out' in a serial fashion by the SMA, pre-SMA, and premotor cortex (Rosenbaum, 1985; Shima and Tanji, 2000). Our finding that sensory attenuation is only affected by the first element in a sequence is consistent with a serial order process that is involved in the preparatory processes preceding a movement sequence. The nature of the information being processed is expressed in real time as the sequence unfolds (Crammond and Kalaska, 2000). This suggests that sensory suppression is associated with the motor and premotor processes involved with the 'unpacking' or sequencing of the individual elements of the motor programme rather than with storage of the programme as a whole (see Figure 9.7). This idea is consistent with previous research (Haggard and Whitford, 2003; Voss et al., 2006; Jiang et al., 1990) which suggests that sensory suppression occurs at the areas where a motor plan is being implemented.

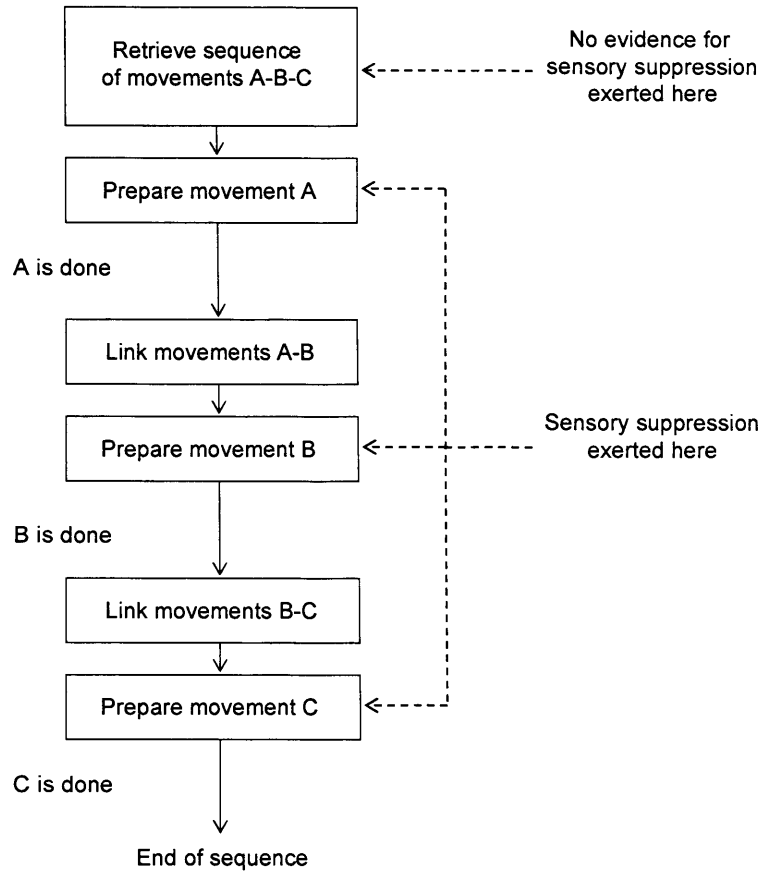

Figure 9.7. Results of Experiment 8 interpreted within the model of Figure 9.1.

The overall picture is one of interacting parallel (buffer) and serial (motor output) processes. At each stage of the cascading process in the frontal executive hierarchy, the precise next action in a sequence is determined by two types of influences: firstly, the processing of the global aspects of the sequence in upper frontal areas and secondly, the sensory signals occurring at the time (Koechlin et al., 2003). The cascading parallel and serial processing of action entails the orderly downward activation of the executive frontal hierarchy.

In cognitive theories of motor control (e.g. Wolpert, 1997; Blakemore et al., 1999), the forward model makes two types of comparisons, one prior to movement and one after movement. First, it predicts the actual outcome of the motor command and compares this to the desired outcome – this comparison occurs *before* a movement is made. This

prediction is used to estimate the state of the system and to make fine adjustments to ongoing motor commands. Second, the forward model predicts the sensory consequences of movement and compares it to the actual sensory feedback - this comparison occurs *after* a movement is made. This prediction can be used to anticipate and compensate for the sensory effects of movement; attenuating the component that is due to self-movement from that due to changes in the outside world. This raises the question as to when a forward model makes these comparisons when presented with a sequence of movements – does it wait for each element in the sequence to be completed before predicting the outcome of the next, or are the predictions for every element made in advance before the sequence is initiated, as in chaining models? The present results suggest that the calculation is made online. The sensory predictions produced in conjunction with the motor command, via an efference copy, seem to gate the perception of the intensity of the self-induced tactile sensations, as appropriate, for each step within a sequence. Thus in terms of the sensory consequences of movement, Sherrington's (1906) classical reflex chaining model is supported, while the advance programming model is not.

Studies with neuropsychological patients are broadly consistent with the concept of a step-by-step running of the motor programme. Patients with Huntington's disease (HD) or Parkinson's disease (PD) were instructed to perform sequential movements by following a squeeze movement as rapidly as possible with a flexion of the elbow (Thompson et al., 1988). They exhibited slowing of the velocity of the movement and marked hesitations between the movement segments compared with controls. Hesitations have been observed by many investigators of HD and PD and are interpreted to reflect a deficit of programme implementations in patients due to disturbed basal ganglia function (Marsden and Obeso, 1994; Weiss et al., 1997). The defects of motor programming seen in both conditions are thought to lie in a dysfunction of the premotor and supplementary motor cortex resulting from abnormal input to these areas from the diseased basal ganglia (Thompson et al., 1988). These patients are not as efficient at binding the elements of a movement together suggesting such binding is one function of the basal-ganglia-frontal motor loop. Based on the current results, sensory suppression also appears to operate on a step-by-step basis, predicting, matching and cancelling just prior to the motor command

for each movement element. The neural basis of sensory suppression would therefore be downstream of the basal-ganglia-frontal binding circuit. Future work could target shocks within the sequence and prior to each element of movement in order to explore this idea further.

A sequence of movements can be rattled out like a chain of responses; the end of one element acting as the stimulus for the initiation of the next. A sequence consists of separate links of elemental movements; sensory suppression precedes each link and only concerns itself with the immediately forthcoming link. Sensory feedback may be packaged separately for each movement, thus allowing the flexible free-flow of everyday behaviour. The successful completion of one step allows the successful implementation of the next. Sensory suppression processes may envelop each step of behaviour informing the motor system at each step: "A is done", "B is done" (Figure 9.7), thereby joining the links of movement into a chain of behaviour.

It should be noted that the sensory suppression effects observed here may only apply to tasks which, such as the current one, are not overlearned. The sequences of finger abductions here were performed at relatively slow production rates. Therefore, the sequence representation may be stored as four distinct elements. However, for overlearned skills such as professional typing, that involve a very rapid sequencing of a large number of different movement elements, the neural systems mediating the sequencing may be represented as a more coherent whole or *chunk* in advance of the movement (Lashley, 1951). Thus, sensory suppression may be affected by *all* of the movements in a "chunked" sequence, rather than just by the first element as observed in the current study. An interesting question for future research is to investigate the role sensory suppression might play in the acquisition and consolidation of new motor synergies.

In conclusion, the main finding of this study is that sensory suppression appears to be only specific to the immediately current movement. We found no evidence that sensory suppression was set in advance for subsequent movements in a sequence. Given the lack

of any sensory suppression effect at the action sequence level, we conclude that sensory suppression operates lower down the motor hierarchy, at the level of movement elements. If sensory suppression is attributed to the forward model of the computational motor control framework (Wolpert, 1997), then our results suggest that forward model predictions are made in real-time, immediately before each movement. Predictions are made just-in-time and are not brought forward to the start of the entire action sequence, even when this is possible.

The final experiment in this thesis (Chapter 10) has a different emphasis. It explores the neural loci of premovement sensory suppression by asking if there is a subcortical contribution to sensory suppression.

Chapter 10: The effects of acoustic startle on sensory suppression

10.1 Abstract

A startling auditory stimulus delivered unexpectedly can activate subcortical structures triggering a prepared movement involuntarily and shortening reaction times. We investigated the effects of the startle acceleration of response on premovement sensory suppression, a phenomenon linked to the voluntary motor command whereby a tactile stimulus is less likely to be perceived on a moving body-part prior to voluntary movement than at rest. Subjects had to detect weak shocks which were delivered to the index finger after a Go signal on some trials. We found that detection rates on movement trials were lower than on non-movement trials, consistent with sensory suppression. In addition, a loud acoustic stimulus was occasionally presented at the same time as the Go signal (startle trials). Reaction times were significantly shorter on startle trials than on other trials, replicating previous startle acceleration of reaction time effects attributed to the operation of subcortical pathways. However, we found no overall difference in premovement sensory suppression effects between baseline and startle movement trials. Rather, startle acceleration of voluntary reactions produced a corresponding acceleration of sensory suppression. Our results provide evidence for a subcortical contribution to sensory suppression and suggest that sensory suppression is a highly general form of motor and sensory interaction.

10.2 Introduction

The startle reaction is considered a vestigial expression of a basic physiological reaction common to all mammals. It is the response to a sudden unexpected stimulus, such as a flash of light or a very loud noise. In human beings, the reaction includes physical movement away from the stimulus, a contraction of the muscles of the arms and legs, and often blinking. It also includes blood pressure, respiration, and breathing changes. The movements that accompany a startle reaction are considered reflex movements under no voluntary control (Valls-Solé et al., 1995).

Muscle response can habituate to a repeated startling stimulus. Habituation is a basic form of non-associative learning involving a decrease in behavioural response to a repeated stimulus (Kupfermann, 1991). Habituation of physiological response has been observed in many studies involving the use of a startling stimulus (Carlsen et al., 2003; Abel et al., 1998; Davis, 1984; Davis and Heninger, 1972; Leaton et al., 1985; Schicatano and Blumenthal, 1998; Valls-Solé et al., 1997). A startling auditory stimulus delivered unexpectedly can reduce the latency of a voluntary response by as much as 77 ms (49%; Valls-Solé et al., 1999). In some subjects, the reaction times can be shorter than the

calculated minimum time for processing of sensory information at the cerebral cortex. Importantly and in contrast to muscle response, startle-accelerated response latency does not habituate over the duration of an experiment (Schicatano and Blumenthal, 1998; Carlsen et al., 2003).

Two other lines of evidence provide further support for the notion of a startle-elicited response. First, while the loud unexpected sound speeds up the execution of a voluntary movement the main characteristics of the movement, as measured by EMG pattern, are not altered. Thus, voluntary reaction time is accelerated while the features of the motor programme are maintained (Valls-Solé et al., 1999). Therefore, the effect of a speeded response is not produced simply by an early startle response superimposing onto a later voluntary response. Second, task accuracy on tasks involving ballistic movement is maintained during the startle-elicited response, suggesting that the response produced at a short latency was indeed the same one that was prepared (Carlsen et al., 2003). The startle response seemed to release a well-formed voluntary action much faster than normal. On this evidence it was suggested that the programme for voluntary action could be stored subcortically, and its rapid output triggered by the startle stimulus.

10.2.1 Subcortical storage

Startle-accelerated response latency depends crucially on preparation. Carlsen et al. (2004a) showed that if a response could be prepared in advance (simple RT) the response was triggered early by startle. However, no RT differences were seen due to startle in a choice RT task (Carlsen et al., 2004b). Thus it appears that a response was triggered by startle only if it was known, and presumably prepared, in advance. Carlsen et al. (2004b) hypothesised that when a response is specified in advance, it may be possible to alleviate cortical demand by offloading sufficient detail of the motor programme to a holding area. Thus, the only information processing requirement would be detection of the imperative stimulus and subsequent triggering of the response. One possible candidate for such motor programme storage is the midbrain reticular formation (Valls-Solé et al., 1999).

Sensory inputs activate the reticular formation and the descending reticulo-spinal tract to the spinal cord (Davis et al., 1982). Because of the differences in the length of the circuits, as well as in the amount of sensory processing, the latencies of the startle reaction are much shorter than those of a voluntary reaction. If this were the case, it may be possible to trigger the motor program in the absence of the normal cortical trigger if some event caused adequate activation of these subcortical storage structures. The very loud acoustic startle stimulus is thought to activate the physiological startle response triggering the stored motor programme at an accelerated latency.

10.2.2 Intersensory facilitation (ISF)

It has been suggested that the RT acceleration effect could be attributed to intersensory facilitation (ISF) or a stimulus-intensity effect (Kohfeld, 1969; Woodworth, 1938) rather than an accelerated motor output. Neural sensory activation due to the acoustic (startle) stimulus might summate with the sensory activation due to a visual (Go) stimulus. The enhanced sensory activation might lead to earlier activation of the normal voluntary motor pathways. However, the amount of shortening due to startle is much greater than that observed in conventional ISF. Intersensory facilitation has been found to shorten reaction time by an amount of 20-50 ms (Nickerson, 1973) whereas the shortening reported by Valls-Solé et al. (1999) was generally more than 70 ms suggesting that some process other than intersensory facilitation is at play.

In sum, the startle stimulus is thought to release aspects of the motor programme stored subcortically in the reticular formation, bypassing the motor cortex (see Figure 10.1). The movement that accompanies a startle reaction is considered to be a reflex movement under no voluntary control but with the same characteristics as voluntary movement (Valls-sole et al., 1995). While physiological startle habituates, response latencies are resistant to habituation.

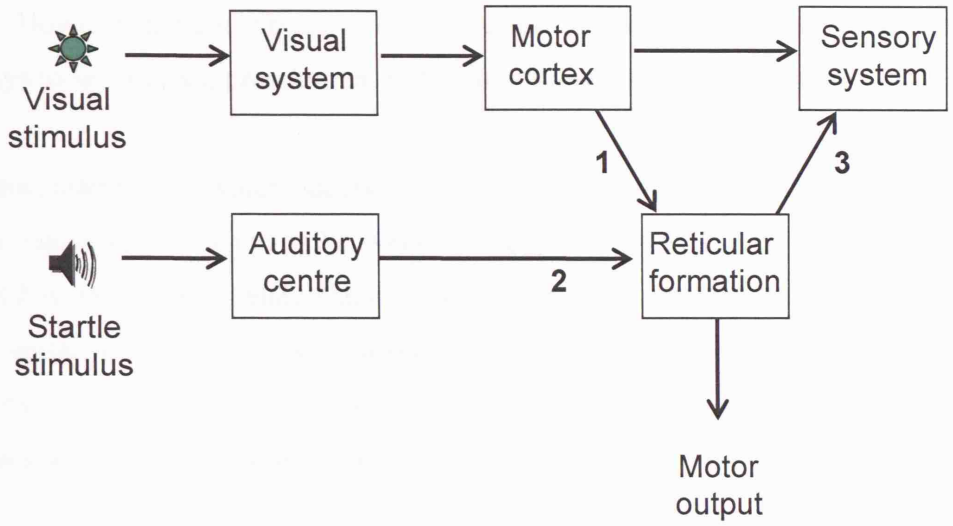

Figure 10.1. A simple model of sensory suppression. During normal conditions the motor output is triggered via the motor cortex (1). In contrast, during a startle trial, a loud acoustic startle stimulus can trigger key aspects of the motor command stored subcortically in the reticular formation, bypassing motor cortex and speeding up reaction times (2). A finding of sensory suppression observed in advance of EMG onset on startle trials would therefore suggest the existence of a direct subcortical-sensory system pathway (3).

10.2.3 Origins of sensory suppression

Evidence for a central origin for premovement sensory suppression has come from studies that show that somatosensory evoked potentials (SEPs) are reduced prior (60-100 ms before the onset of EMG activity) to voluntary movement but not passive movement (Ghez and Lenzi, 1971; Chapman et al., 1988) suggesting that sensory suppression depends on the *volitional* nature of the motor act. To test whether sensory suppression relies on central signals related to the preparation for movement, Voss et al. (2006) used transcranial magnetic stimulation (TMS) over primary motor cortex to delay the output of motor commands from the motor cortex during voluntary movement. They found that sensation was attenuated during the delay period, in the absence of movement. They concluded that cortical stages of the motor hierarchy which prepare motor commands contribute to the production of sensory suppression of voluntary movements. This evidence suggests that sensory suppression that occurs in advance of normal voluntary movement is central in origin and arises, at least in part, upstream of primary motor

cortex. However the contribution of other central motor structures and subcortical motor pathways to sensory suppression has not, to our knowledge, been tested directly.

Of course, attenuation which occurs at one site will also produce effects at higher areas in sensory pathways. For example, while the results from SEP studies (Ghez and Lenzi, 1971; Chapman et al., 1988) show evidence for premovement sensory suppression in central areas, they do not prove that sensory transmission is modified at central synapses, since any sensory gating that is occurring at the spinal level would also change cortical responses to peripheral stimuli (Seki et al., 2003).

Therefore, while premovement sensory suppression is thought to depend upon the voluntary nature of the motor act and to be cortically-mediated, movement that is fast-tracked by a startle stimulus is thought to involve reflexive, involuntary mediation by subcortical structures (Carlsen et al., 2004a). If sensory suppression depends only on the cortically-mediated, volitional nature of the movement, we might reasonably predict that for involuntary startle trials sensory suppression will not be brought forward in time with the movement to reflect subcortically-mediated, startle acceleration of reaction time. In this study, subjects were required to perform the S-D (sensory-detection) task reporting the presence of a weak electrical cutaneous shock that was delivered on some trials. Occasionally, the Go signal was presented simultaneously with an acoustic startle stimulus. The difference between shock detection rates applied to the moving finger and the finger at rest served as a measure of sensory suppression. Premovement central suppression was obtained by confining the analysis to the detection of shocks delivered prior to EMG onset.

A comparison of sensory detection rates and their time-courses on baseline and startle movement trials allowed us to investigate whether sensory suppression effects are tied to the involuntary startle-accelerated movement in the same way as to voluntary movement. If the signal driving the sensory suppression effect is purely cortical (consistent with premotor accounts) then we predict that sensory suppression prior to movement in voluntary movement trials (Figure 10.2a) but no sensory suppression prior to startle-

accelerated trials (Figure 10.2b). In contrast, consistent sensory suppression effects occurring in advance of EMG onset across both baseline and startle movement trials (Figure 10.2a and 10.2c) would imply that subcortical motor circuits responsible for accelerated startle induced output of voluntary reactions also have access to the cortical centres responsible for sensory detection. A main experiment and a control experiment comparing these predictions are presented.

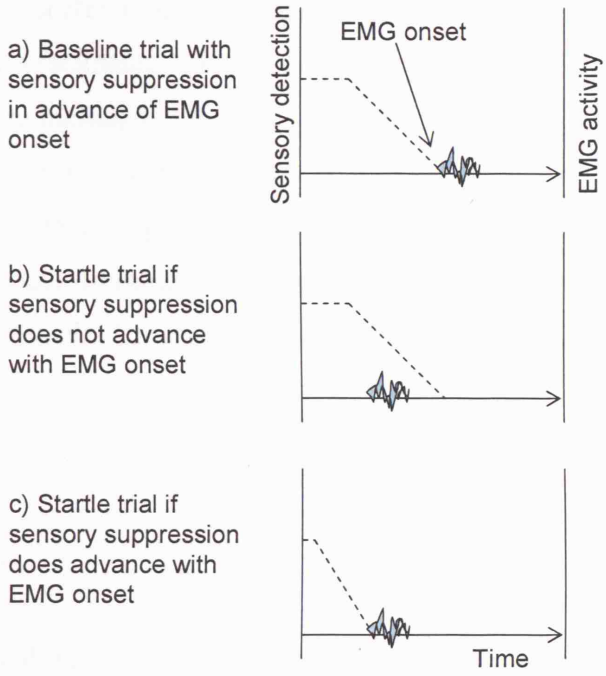

Figure 10.2. Schematic of possible interaction between startle reaction and sensory suppression. a) Sensory detection (dashed line) normally decreases in a time-dependent manner prior to onset of EMG during a voluntary baseline trial. b) During a startle trial, one prediction is that sensory suppression will not be tied to EMG onset when the movement is accelerated. However, c) if sensory suppression shifts with the accelerated movement and remains tied to EMG onset, then this would suggest the existence of a projection from subcortical motor pathways to the sensory system (see line "3"; Figure 10.1).

10.3 Materials and Methods

10.3.1 Subjects

Sixteen paid subjects took part with local ethical committee approval. The data from 3 subjects were excluded because they did not show sufficient startle acceleration of reaction time. Typically, the orbicularis oculi (OOc) or sternocleidomastoid (SCM) muscles are used to assess the startle response (e.g. Kumru and Valls-Solé, 2006). Here we use reaction time acceleration as a measure of the startle reaction. Only data from subjects who showed a facilitated reaction time i.e. a startle RT equal to 75% or less than their baseline RT, were included in the analysis. This criterion was based on results from a pilot study (not reported here) in order to produce accelerated startle RT effects consistent with other studies (e.g. Sanegre et al., 2004; Carlsen et al., 2003; Kumru and Valls-Solé, 2006). Data from the remaining 13 subjects were included in the final analysis; all but 4 were male, 12 were right-handed and the mean age was 24.4 (SD = 4.1) years.

10.3.2 Procedure

The subject's right hand was positioned with the index finger resting on the pivoting plate (Figure 10.3). A simple staircase procedure (Levitt, 1971) first established the shock intensity at which approximately 90% of stimuli delivered to the resting finger were detected. Perceived intensity was varied by adjusting stimulator pulse-width. This level was then used throughout the experiment. Occasionally, it was necessary to fine-tune the intensity level of the stimulus during a practice block. Stimulus intensity then remained constant throughout the experimental blocks. The staircase procedure was repeated at the end of the experiment.

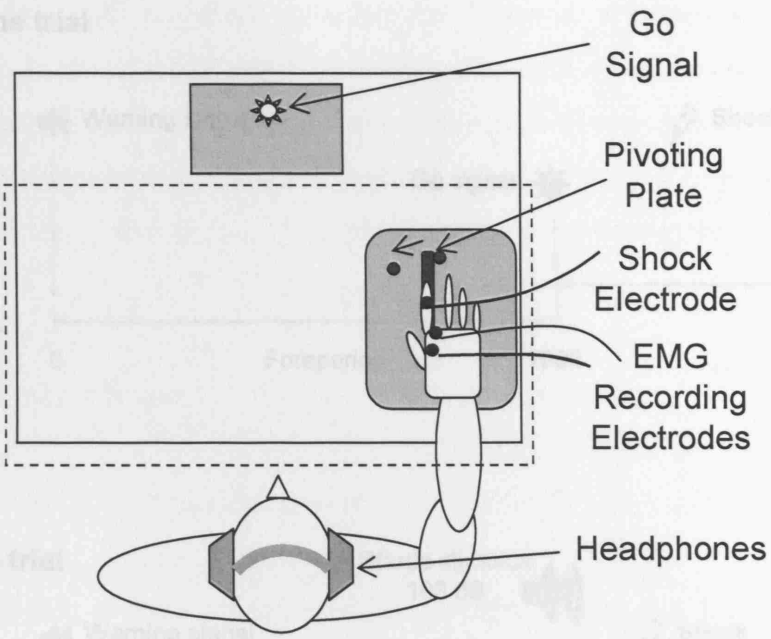

Figure 10.3 Experimental set-up. Subjects placed their right index finger on a pivoting plate. The Go signal was presented via an LED in front of the subject. Direct vision of the hand was prevented. The startle stimulus was presented binaurally via headphones.

The prior instruction procedure (see e.g. Experiment 9) was repeated. A verbal instruction "move" or "don't move" was given by the experimenter at the start of each trial. See Figure 10.4 for trial design. The start of each trial was marked by an acoustic signal (35 dB). After each trial, subjects reported verbally ('yes'/'no') whether they perceived a shock stimulus. The experimenter initiated each trial, ensuring an intertrial interval of at least 3 seconds. No feedback was given.

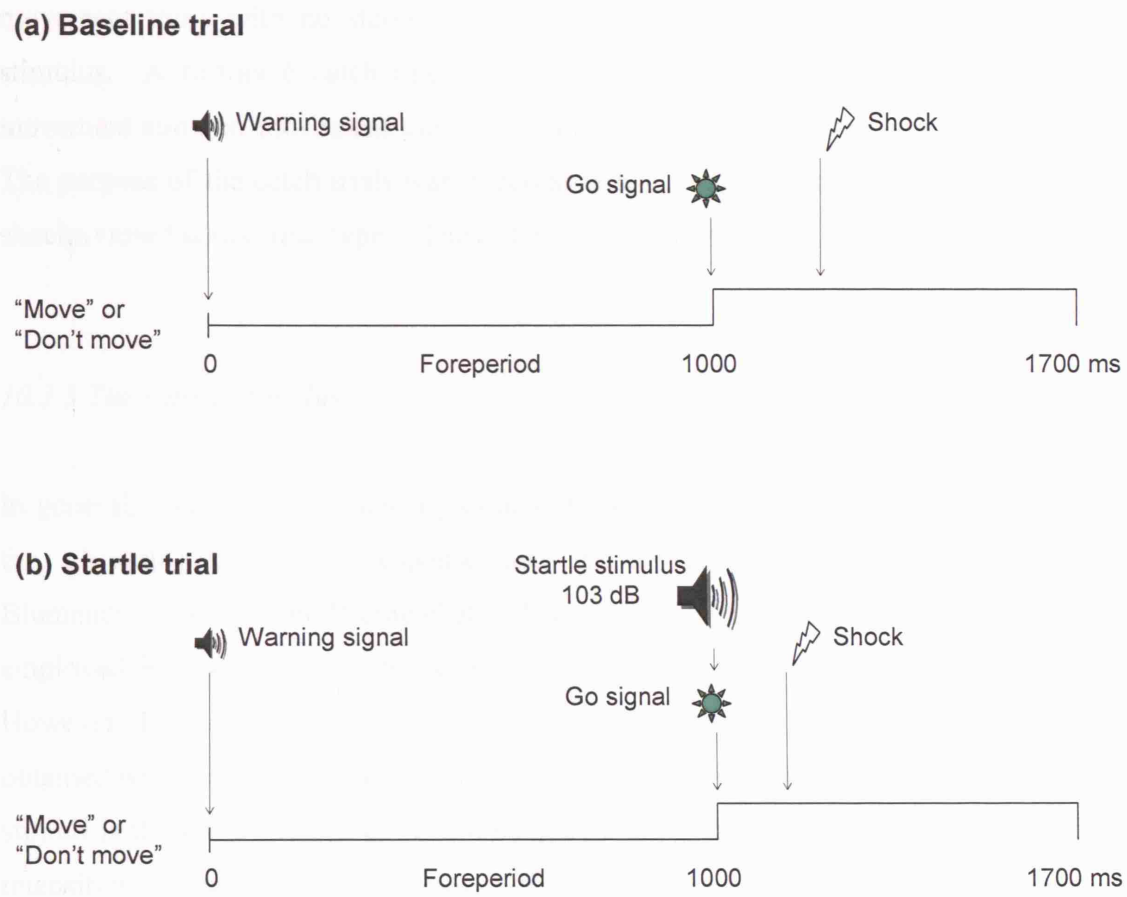

Figure 10.4. Experimental design. (a) The experimenter gave an instruction at the start of each trial, either 'move' or 'don't move'. After an acoustic warning signal (35 dB), the Go signal was presented after 1000 ms. Subjects either moved or did not move their index finger during the 700 ms response window. The shock was delivered 50 ms prior to each subject's mean reaction time. (b) On startle trials, the very loud acoustic stimulus (103 dB) was delivered simultaneously with the Go signal. The shock was delivered 50 ms prior to each subject's mean startle-accelerated reaction time.

Subjects performed a practice block of 44 trials prior to the experimental blocks in order to become familiar with the task and the equipment. Instructions emphasised fast reaction times and consistent movements. It was made clear that no acoustic startle stimulus would be presented during this block. Practice was followed by 4 experimental blocks. Subjects were warned that some trials would contain a loud irrelevant stimulus (the startle stimulus) and were instructed to respond to the Go signal as per the prior instruction ("move" or "don't move"). Each block consisted of 54 trials comprised of 27 movement trials with no startle stimulus, 9 movement trials with the startle stimulus, 9 non-

movement trials with no startle stimulus and 3 non-movement trials with the startle stimulus. A further 6 catch trials (no shock stimulus) were divided equally between movement and non-movement trials. Two of the catch trials contained a startle stimulus. The purpose of the catch trials was to access whether the subject's criterion for detecting shocks varied across trial types. The order of the trials was randomised.

10.3.3 The startle stimulus

In general, increasing the intensity of acoustic startle stimuli has the effect of increasing the physiological startle response and decreasing response onset latency. (e.g. Blumenthal, 1988; 1996; Blumenthal and Berg, 1986). Many acoustic startle studies have employed intensities in the range of 100 dB SPL or more (Blumenthal et al., 2005). However, Blumenthal and Goode (1991) demonstrated that startle responses could be obtained with stimuli in the range of 50 to 70 dB SPL. An advantage of using less intense stimuli is the minimization of risk to subjects from unnecessarily high acoustic stimulus intensities (see "The Control of Noise at Work Regulations 2005"; www.hse.gov.uk/noise/regulations.htm for details). Startle responses are also influenced by stimulus rise time, a measure of how quickly the stimulus reaches its full, steady-state amplitude. Startle stimuli with shorter rise times elicit responses with shorter onset latency, presumably because startle is specialised for the detection of sudden changes in the environment (Blumenthal and Berg, 1986; Graham, 1992). The duration of the stimulus also affects startle responding. Longer duration stimuli, up to approximately 50 ms, are associated with larger response magnitude and amplitude, and higher response probability and is considered sufficient for startle elicitation (Putnam and Roth, 1990). Therefore, a stimulus of 50 ms duration (rise time < 1ms; frequency = 1500 Hz) and a sound level of 103 dB was chosen as a compromise between subject comfort and safety and startle effectiveness. A pilot study (not reported here) demonstrated that this acoustic stimulus produced significant acceleration of startle response. The very loud sound produced in this way was calibrated with a Brüel and Kjaer precision sound level meter type 2203. The sound was created using a gated tone generator (custom-built by UCL

Institute of Neurology, Sobell Research Department). The startle stimulus was delivered binaurally via headphones. Subjects were allowed to hear the startle stimulus a few times before commencing the experiment. Care was taken when introducing the subjects to the loud noise for the first time to avoid discomfort.

10.4. Results; main experiment

Figure 10.5 shows an illustration of a typical baseline and startle trial. Note the faster reaction time for the startle trial relative to the baseline trial and the similar profiles of the EMG burst and movement kinematics for each trial type.

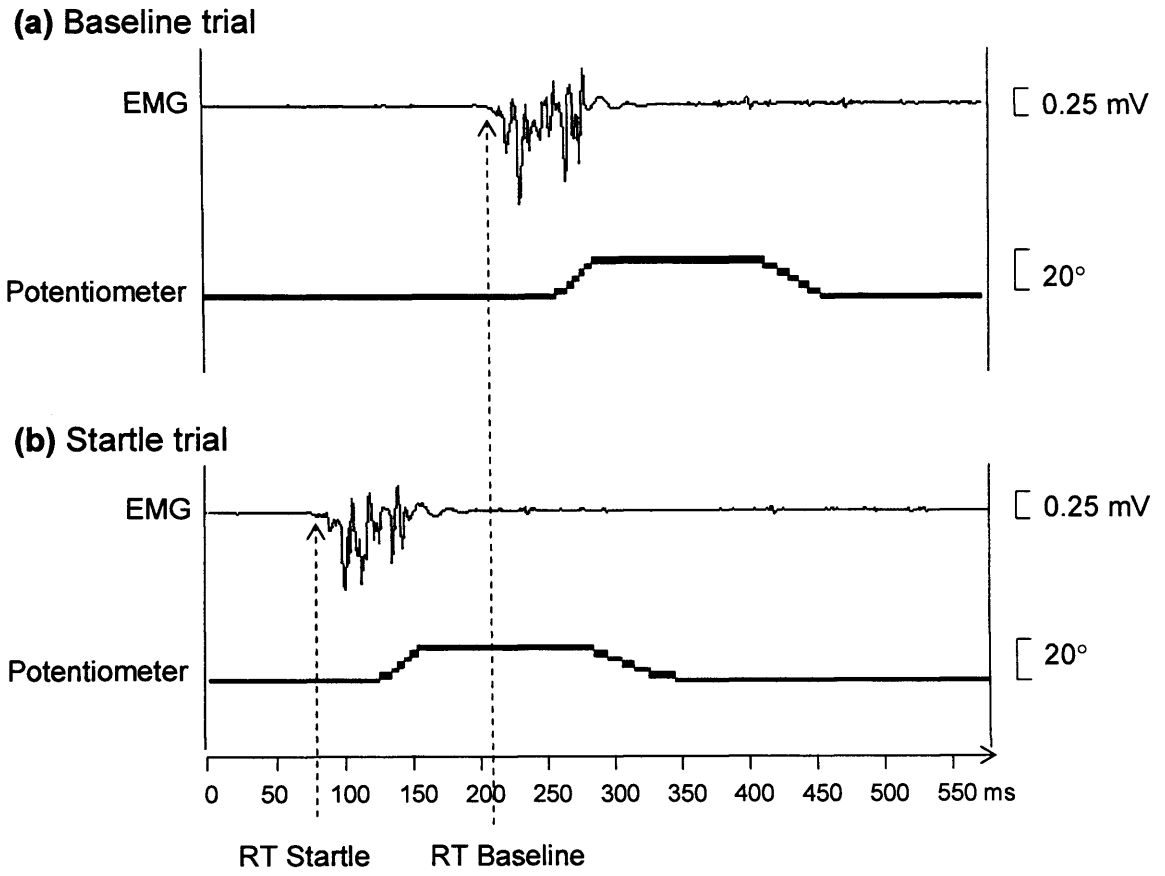

Figure 10.5. Illustrative trials showing EMG recordings and movement potentiometer output for a single subject. Reaction time was defined as the onset of EMG. a) Baseline trial in which the subject responded to the visual Go signal. b) Startle trial in which the Go signal was presented simultaneously with the startle stimulus. Note the shortened reaction time (RT) with the startle stimulus.

On catch trials (no shock stimulus) only 2.9% false positive detections were recorded, indicating that subjects used a very conservative response strategy. A 2 x 2 ANOVA carried out on the number of false positives showed that there was no effect of movement (movement vs. non-movement) $F(1,12)=0.649$; $p=0.436$, nor trial type (baseline vs. startle) $F(1,12)=0.381$; $p=0.549$ for baseline and startle trials (see Table 10.1). This suggests that the startle stimulus did not influence the criterion for shock detection.

Table 10.1. The number of false positive catch trials pooled across subjects during movement and non-movement trials for baseline and startle trials.

	Trial Type	
	Baseline	**Startle**
Movement	1	4
Non-movement	2	1

Errors of commission (i.e. when a non-movement trial was accompanied by EMG activity) were 0.8% on baseline trials and 0.6% on startle trials. Errors of omission (i.e. movement trials without movement during the response window) occurred on 0.7% of all movement trials. These trials were excluded when measuring the effects of sensory suppression. The pre- and post-experiment staircases showed similar shock intensity thresholds (mean pulse-widths = 31 and 30 μs respectively). These values did not differ significantly $t(12)=0.984$; $p=0.345$.

10.4.1 Reaction times

Reaction times for each subject were trimmed to +/- 2 SD (excluding 4.8% of baseline movement trials and 6.6% of startle movement trials) and subjected to a one-way ANOVA. We observed the expected acceleration of reaction time on startle trials. The mean reaction times for baseline and startle movement trials were 201 ms and 128 ms respectively, yielding a shortening of reaction time of 73 ms. This difference was significant $t(12)=8.326$; $p<0.0001$ and corresponds to a mean startle reaction time that is 64% of the mean baseline reaction time, consistent with other startle studies [e.g. Sanegre et al., 2004 (69%); Carlsen et al., 2003 (77%); Kumru and Valls-Solé, 2006 (64%)].

10.4.2 Shock detection

As the extent of sensory suppression varies with the temporal relation between stimulus and movement beginning up to 120 ms before EMG onset (Williams et al., 1998), it is critical that the timing of the shock across baseline and startle movement trials is similar so as to be able to compare sensory suppression rates. The mean duration between shock and EMG activity did not differ significantly between each type of trial $t(12)=0.569$; $p=0.580$, suggesting that our adjustment of shock to each subject's mean RT in the practice block was successful.

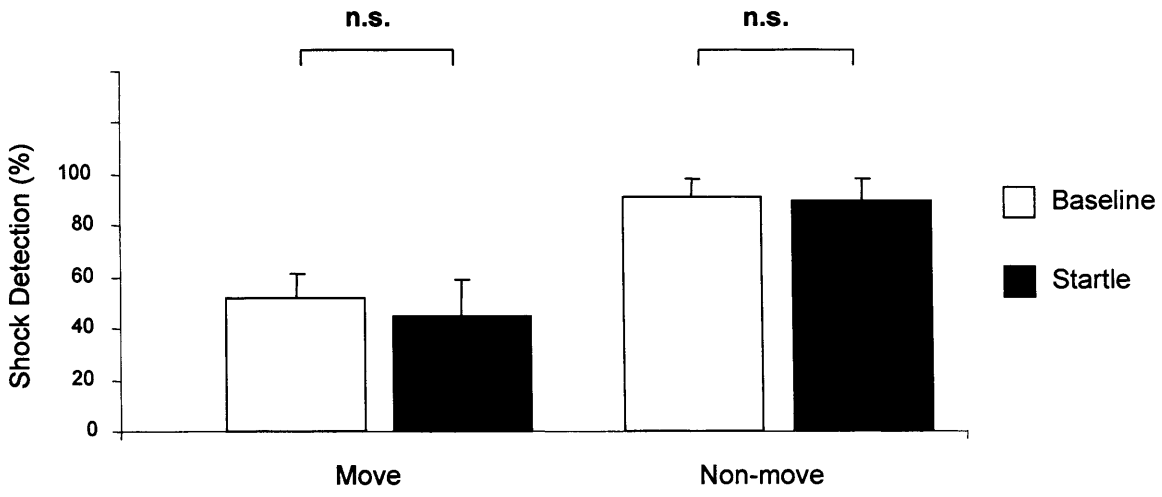

Figure 10.6. Main experiment (N=13); the mean percentage of electrical stimuli detected (+/- SD) for baseline and startle trials during movement and non-movement trials.

Figure 10.6 shows shock detection rates for each trial type. A repeated measures ANOVA showed a significant main effect of movement $F(1,12)=74.892$; $p<0.0001$, no main effect of trial type $F(1,12)=1.008$; $p=0.335$, and no significant interaction between movement and trial type $F(1,12)=0.934$; $p=0.353$.

10.4.3 Stability across time of perceptual performance at rest.

In order to study the critical interval of sensory suppression just prior to EMG onset, trials occurring from 150 ms before and up to 50 ms after EMG onset were grouped into 50 ms bins. The percentage of shocks detected was calculated for each bin and the graph plotted (see Figure 10.7) in order to show the temporal evolution of perceptual abilities relative to EMG onset. In order to study the key interval of 120 ms before EMG onset (Williams et al., 1998) the curves were compared for the three bins within this period i.e. -100, -50 and 0 ms relative to EMG onset. T tests revealed an unexpected, non-significant tendency for *greater* premovement sensory suppression for startle trials, although the hypothesis outline (Figure 10.2) predicts less suppression for startle trials, relative to baseline trials for the two bins 100 ms $t(12) = 0.951$; $p=0.360$ and 50 ms before EMG onset $t(12) = 1.889$; $p=0.083$ (both two-tailed). At EMG onset, sensory suppression effects were in the predicted direction with less sensory suppression for startle than for baseline trials; however this difference was also not significant $t(12) = 0.246$; $p=0.398$ (one-tailed).

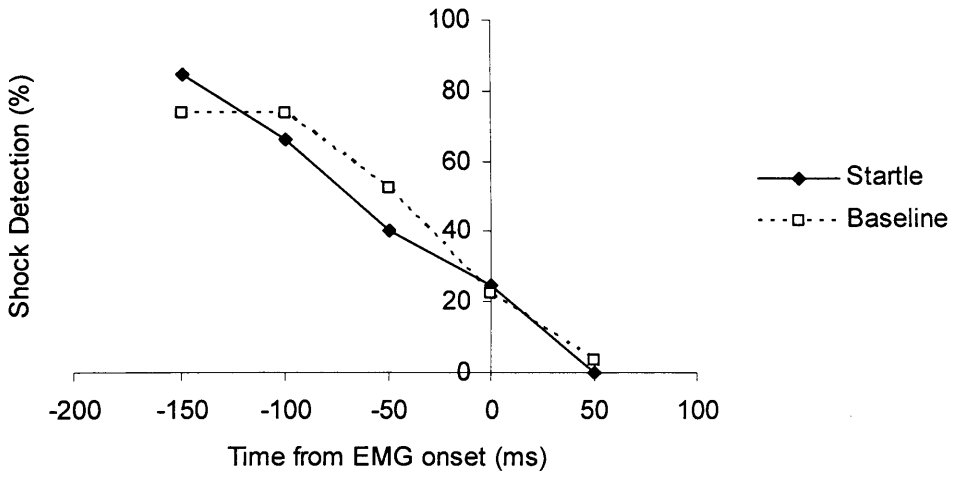

Figure 10.7. Effects of index finger abduction on the detection of fixed intensity stimuli applied to the moving finger for startle and baseline movement trials (50 ms bins). Detection performance over time is plotted relative to the onset of EMG (0 ms).

10.4.4 "Pure" startle trials

It was of interest to examine startle trials with reaction times of less than 100 ms (mean RT = 83 ms; range 55 – 99 ms) for two reasons. Firstly, these trials are considered to be "pure" startle trials since they are clearly faster than any possible voluntary reaction (Valls-Solé et al., 1999). Secondly, it is not known if sensory suppression occurs when reaction times are strongly accelerated. Therefore the data were re-sorted into 20 ms resolution bins in order to adequately examine the sensory suppression curve for startle trials with RTs less than 100 ms (see Figure 10.8). For the earliest time-bin, 40 ms before EMG onset i.e. 43 ms earlier than the mean RT of 83 ms of these pure startle trials, mean shock detection rates were 35% during movement trials and 91% during non-movement trials; $t(3)=3.316$; $p=0.023$ (one-tailed). These percentages are the means from those 4 subjects who contributed sufficient data-points to this bin to estimate percentage values (i.e. at least 4 trials per subject, average of 7 per subject, total of 31 trials overall). This subset of trials shows that sensory suppression activates quickly (in 43 ms or less after the Go signal) and powerfully, dampening shock detection rates from 91% to 35% in this interval. Sensory suppression effects persevered for the time-bin 20 ms before EMG onset (at least 4 trials per subject, average of 6 per subject, total of 34 trials overall). Mean shock detection rates were 30% during movement trials and 88% during non-movement trials; $t(4)=4.514$; $p=0.005$ (one-tailed).

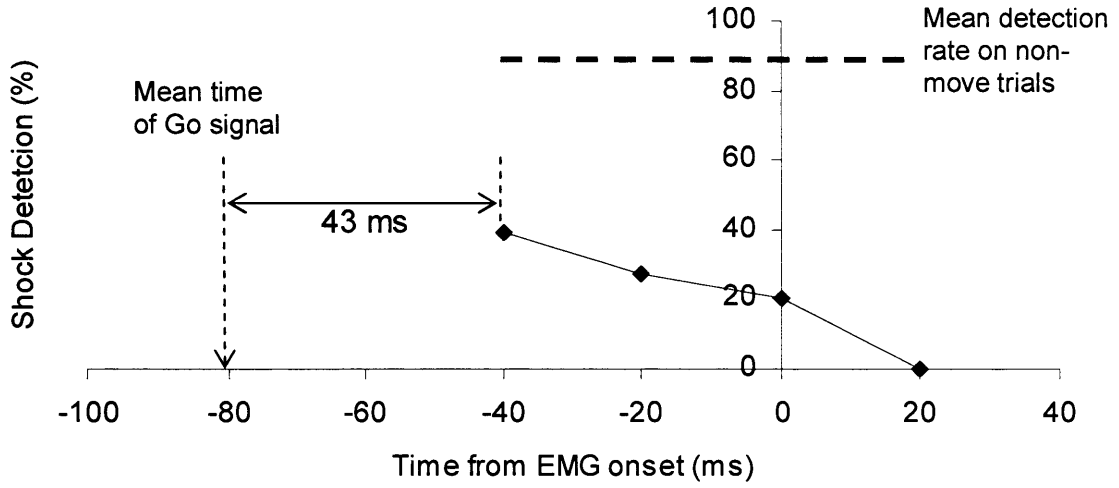

Figure 10.8. Effects of index finger abduction on the detection of fixed intensity stimuli applied to the moving finger for "pure startle" trials with reaction times less than 100 ms (mean RT = 83 ms). Detection performance is plotted in 20 ms bins relative to the onset of EMG (0 ms). The dashed vertical arrow shows mean time of Go signal, 83 ms before EMG onset.

In the main experiment, the foreperiod prior to the Go signal in each trial was fixed (1000 ms). Subjects may have tended to anticipate the Go signal and respond early or even in advance of the signal. This could have resulted in some trials with reaction times that were artificially fast, in turn leading to an underestimation of startle acceleration of RT. We therefore performed a short control experiment with less predictable Go signals to ensure that our results reflected genuine reactions rather than anticipations.

10.5 Control experiment

10.5.1 Subjects

Six naïve paid subjects took part with ethical committee approval. The data from 2 subjects were excluded because they did not show sufficient startle acceleration of reaction time (< 25% of baseline RT, as in the main Experiment). Data from the remaining 4 subjects were included in the final analysis; 3 were female, all were right-handed and the mean age was 24.8 (SD=3.9) years.

10.5.2 Procedure

All procedures were identical to the main Experiment outlined above with the following exception. The acoustic warning signal at the start of each trial was followed by a variable foreperiod of either 900, 1000 or 1100 ms presented randomly within a block. During startle trials as in the main experiment, the startle stimulus was always presented simultaneously with the Go signal.

10.6 Results; control experiment

On catch trials only 1.0% false positive detections were recorded. There was no difference in the number of false positives on catch trials between baseline and startle trials (p=0.391). There were no errors of commission (i.e. when a non-movement trial was accompanied by EMG activity) on either baseline or startle trials. Errors of omission (i.e. movement trials without movement during the response window) occurred on 1.1% of all movement trials. These trials were removed from the analysis. The pre- and post-experiment staircases showed similar shock intensity thresholds (mean pulse-widths = 34 and 33 μs respectively). These values did not differ significantly, t(3)=1.213; p=0.312.

10.6.1 Reaction times

Reaction times for each subject were trimmed to +/- 2 SD (thereby excluding 6.3% of baseline movement and startle movement trials) and subjected to a one-way ANOVA. As in the main experiment, we observed the expected shortening of reaction times on startle trials. The mean RTs were slightly longer than in the main experiment: 247 ms in baseline trials and 158 ms in startle trials. This confirms the possibility that some anticipation may have occurred in the main experiment. However, the startle acceleration of reaction time was equally present in the control experiment. The shortening of reaction time was 89 ms, corresponding to a mean startle reaction time that is 64% of the mean baseline reaction time. This difference was significant t(3)=10.419; p=0.002. The mean interval between shock and EMG activity did not differ significantly across trial types t(3)=0.1888; p=0.156, confirming that our adjustment of shock timing to each subject's mean RT in each condition was successful.

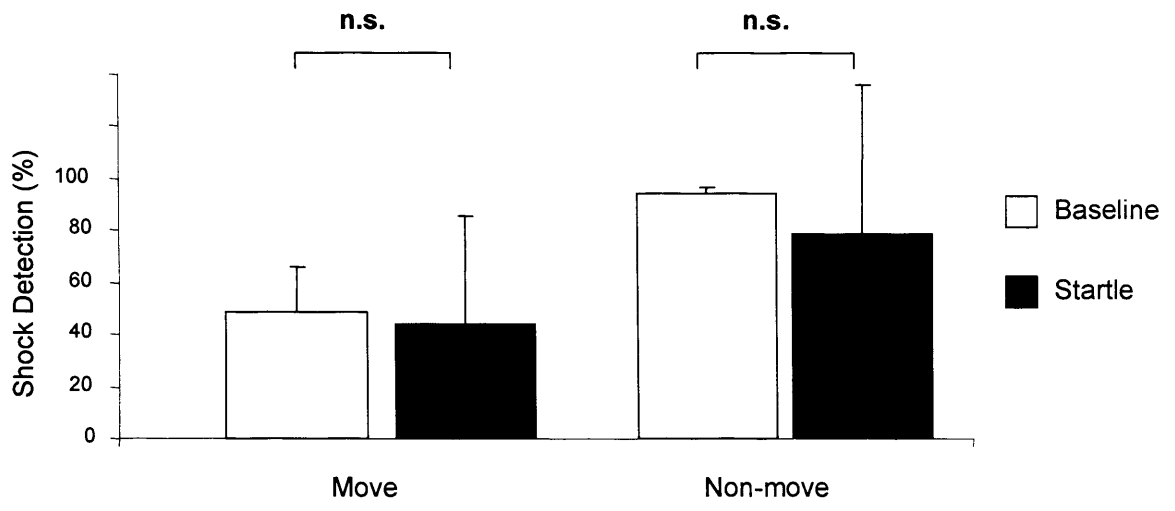

Figure 10.9. Control Experiment (N=4); the mean percentage of electrical stimuli detected (+/- SD) for baseline and startle trials during movement and non-movement trials. The high errors bars in the startle trials are due to one subject who detected relatively few shocks during startle trials but nevertheless showed a difference between detection on move and non-move trials similar to the other subjects.

10.6.2 Shock Detection

Shock detection rates for the baseline movement and non-movement trials were 49% and 94% respectively. On startle trials shock detection rates were 44% for movement trials and 79% for non-movement trials (see Figure 10.9). A repeated measures 2 x 2 ANOVA showed a significant main effect of movement $F(1,3)=58.270$; $p=0.005$, no main effect of trial type $F(1,3)=0.519$; $p=0.523$, and no significant interaction between movement and trial type $F(1,3)=0.557$; $p=0.510$. Thus, the control experiment showed the same startle induced acceleration of the sensory suppression process as in the main experiment.

10.7 Discussion

We used an acoustic stimulus previously demonstrated to evoke the robust startle-induced acceleration of reaction time that is indicative of subcortically-mediated movements (e.g. Sanegre et al., 2004). Furthermore, we replicated premovement sensory suppression effects for voluntary movements as previously demonstrated by Williams et al. (1998) and extended these effects to startle movements with quickly accelerated reaction times. Both voluntary (cortical) and involuntary (subcortical) initiated actions produced suppression of afferent sensation from the moving body part. This finding suggests that the subcortical structures governing fast, automatic motor output also have access to the same circuits that are involved in movement-related modulation of sensations.

Voss et al. (2006) demonstrated that sensory suppression relies on central signals related to the preparation for movement and that these signals arise upstream of primary motor cortex. They suggested a cortical, premotor origin of the sensory suppression signal. A similar conclusion was reached by Haggard and Whitford (2004). In contrast, the present results suggest an important, additional *subcortical* contribution to sensory suppression. Rapid startle-accelerated motor reactions are widely held to involve a subcortical motor pathway (Valls-Solé et al., 1999; Sanegre et al., 2004; Carlsen et al., 2004a). We showed that such fast motor reactions can produce comparable premovement sensory suppression to voluntary reactions. This implies that subcortical motor structures can send fast signals to attenuate activity in appropriate centres on the afferent somatosensory pathway.

Considering the present results in conjunction with previous reports of premotor sensory suppression (Voss et al., 2006; Haggard and Whitford, 2004), we suggest there may be *several* origins of sensory suppression signals at multiple levels of the descending motor pathways. In addition, it is known that sensory attenuation can occur at several locations on the afferent somatosensory pathway. These multiple sensory suppression signals could also reach several destinations. The locations where sensory attenuation may occur include the spinal cord (Seki et al., 2003), dorsal-column nuclei (e.g. Gordon and Jukes, 1964) and somatosensory cortex (e.g. Rushton et al., 1981). Our results can suggest a

subcortical origin of a suppression signal, but cannot distinguish whether the destination of this signal is cortical or spinal (see Figure 10.10).

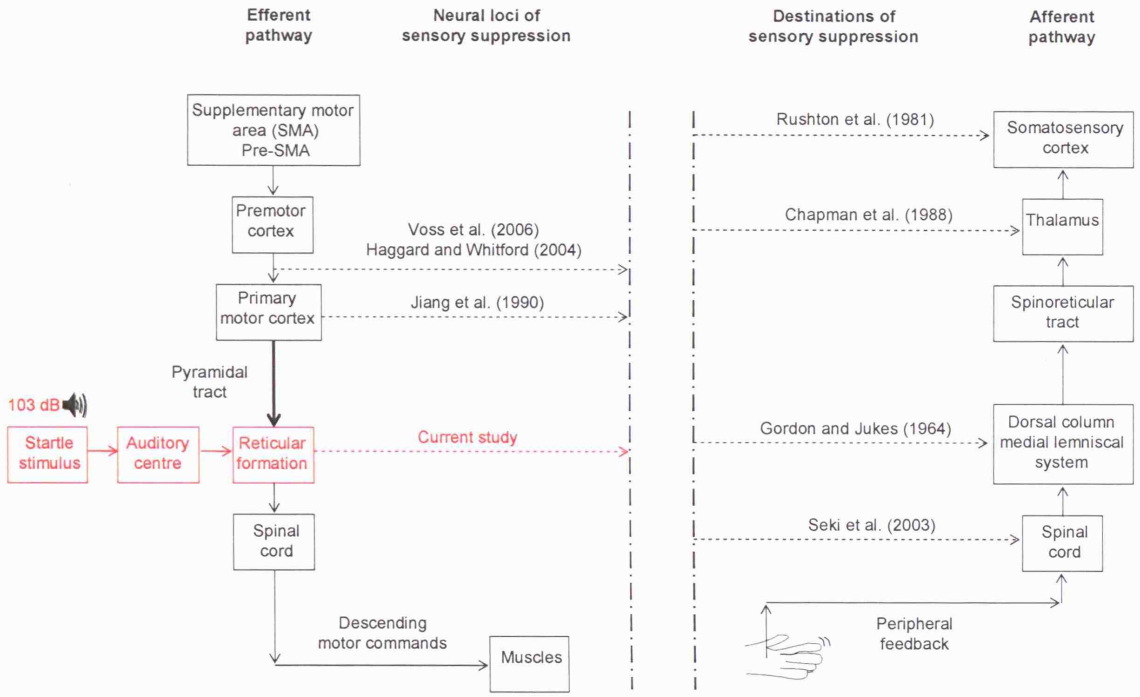

Figure 10.10. Schematic of efferent (signals causing sensory suppression) and afferent (locations where attenuation occurs) pathways showing some possible interactions (dashed arrows). The current results (shown in red; Walsh and Haggard, 2008) provide evidence for a subcortical contribution to sensory suppression.

Furthermore, we separately analysed all movement startle trials with a reaction time of 100 ms or less. There were two reasons for doing this. Firstly, these faster reaction times may be considered "pure" startle movements as the very short reaction times (mean RT = 83 ms) make processing of sensory information at the cerebral cortex more unlikely due to neural restraints on transmission time (Valls-Solé et al., 1999). Secondly, movement trials with a reaction time of 100 ms or less raise an interesting question from a sensory suppression perspective. In a simple RT task with a mean reaction time of 250 ms, sensory suppression processes typically arise 120 ms in advance of the onset of EMG

activity during voluntary movement (Williams et al., 1998). If the reaction time is strongly shortened by the startle stimulus, yet premotor sensory suppression is still found, then the resulting values can be used to estimate the temporal response of the neural systems responsible for sensory suppression. Our results show that sensory suppression consistently begins in advance of movement even when these movements are strongly accelerated by startle stimuli. We found that sensory detection on pure startle trials was reduced from 91% to 35% within 43 ms of a combined Go and startle stimulus. This suggests that the modulation of sensory pathways by putative subcortical motor structures is both rapid and powerful. However, our results cannot directly reveal the relative importance of premotor (e.g. Voss et al., 2006) and subcortical (present study) pathways to sensory suppression. Future research could compare and quantify the respective strengths of both cortical and subcortical inputs to sensory suppression.

Startle-induced shortening of reaction times has also been attributed to intersensory facilitation (ISF; Nickerson, 1973) rather than accelerated motor output. According to this theory, neural sensory activation due to an acoustic (startle) stimulus might summate with the sensory activation due to a visual (Go) stimulus. The enhanced sensory activation might lead to earlier activation of the normal voluntary motor pathways. Thus, intersensory facilitation would produce faster reaction times in startle than in baseline trials for purely sensory reasons. Previous studies have considered intersensory facilitation as a possible alternative explanation of startle-induced acceleration of voluntary reaction times (e.g. Carlsen et al., 2004b), but have rarely been able to rule it out entirely. In contrast, the ISF hypothesis makes a clear prediction for our data. If the startle stimulus indeed facilitates sensory processing, it should presumably lead to facilitation of shocks as well as processing of visual Go signals. Acoustic-somatosensory facilitation can be stronger than acoustic-visual facilitation (see Figure 10.18 in Stein and Meredith, 1993). Our data showed no evidence of improved somatosensory detection in the presence of startle stimuli, and indeed we found a trend in the opposite direction (Figure 10.7). Therefore, our results also provide important evidence against an intersensory facilitation interpretation of startle acceleration, and thus in favour of a motor interpretation.

Theories of computational motor control (e.g. Wolpert, 1997; Blakemore et al., 1999; Shergill et al., 2003) suggest that feedback is a very important component of sensory suppression. The forward model makes predictions of the sensory feedback based on the motor commands. These predictions are then compared to the estimated sensory feedback to produce the sensory prediction errors or sensory discrepancy. When the actual and the predicted sensory feedback match, they cancel and sensory information is attenuated. Presumably on startle trials, there is no time for feedback (Carlsen et al., 2003); yet sensory suppression was still observed. The current results, where sensory suppression is observed despite acceleration of reaction times, present feedback accounts of sensory attenuation (e.g. Wolpert, 1997; Blakemore et al., 1999) with a challenge to explain how sensory suppression can take place in the absence of feedback, and indeed substantially earlier than feedback would be predicted to arrive.

Involuntary muscle movements may be triggered by single-pulse transcranial magnetic stimulation (TMS) of the primary motor cortex. An interesting question is whether such movements give rise to premovement sensory suppression. According to computational motor control theories, if the somatosensory consequences of TMS-induced movement are predictable, then the efference copy of the movement should be cancelled resulting in sensation that is attenuated (e.g. Wolpert, 1997). Chronicle and Glover (2003) tested this hypothesis by adopting Blakemore's tickle procedure (see Section 1.5.2; Blakemore et al., 1999). They investigated whether a stimulus applied to the sole of the foot by a TMS-induced involuntary movement of the left hand, would be perceived as ticklish. They found that involuntary self-stimulation of the foot consequent upon TMS of the motor cortex was rated just as ticklish as external, experimenter-produced stimulation. The relative lack of attenuation of sensation during TMS suggests that magnetic stimulation of the motor cortex does not give rise to an efference copy of motor output. Voss et al. (2006) reached a similar conclusion: they found normal sensory suppression during the delay prior to a movement caused by TMS over the motor cortex in a reaction time task. Both studies suggest that sensory suppression in normal circumstances reflects central signals related to the preparation for movement, and these signals are *upstream* of primary motor cortex. Haggard and Whitford (2004), for example, suggested a source for

these signals in the SMA. However, the details of the circuitry are unclear. It is known that premotor areas project to intralaminar regions of the thalamus and are involved in motor-sensory integration (Macchi and Bentivoglio, 1986). Therefore, TMS applied to upstream premotor regions, but not primary motor cortex, could, in principle, induce an efference copy and consequent sensory attenuation. This prediction could be tested in subsequent research.

In conclusion, an acoustic startle stimulus shortened reaction times in a manner consistent with activation of subcortical motor pathways. Even under these conditions, consistent sensory suppression processes were observed. The process of sensorimotor attenuation appears to operate regardless of the voluntary (cortical) or involuntary (subcortical) nature of the movement being made. There are multiple possible sources of sensory suppression. Equally, there are multiple possible destinations of sensory suppression in the afferent somatosensory pathway. Our data show that a subcortical origin exists as well as the premotor origin previously described (Voss et al., 2006; Haggard and Whitford, 2004). Therefore, sensory suppression appears to be a general form of motor to sensory interaction rather than a single, specific physiological circuit.

Chapter 11: General discussion and conclusions

Movement-related sensory suppression is an example of a motor-sensory interaction whereby sensory stimuli are perceived as less intense before and during a movement than at rest. This thesis focused on sensory suppression of a weak electrocutaneous stimulus delivered just prior to movement onset, and developed this situation as a paradigm for studying motor-sensory interactions. A brief summary of the main findings of the experiments reported in this thesis is first presented. This is followed by a description of some ways in which this thesis contributes to current knowledge.

First, it was shown that sensory suppression occurred for actions which were prepared (Chapter 2), but then inhibited before execution (Chapter 3). When the response is known in advance, sensory suppression can also occur during the preparatory foreperiod of a trial, prior to the Go signal (Chapter 4). The recovery from sensory suppression after successful inhibition of a movement offers a new way of measuring the processes triggered by a NoGo signal. Thus, it was shown that "going" and "stopping" have distinct sensory signatures (Chapter 5). However, under certain circumstances these sensory signatures can dissociate. For example, when Go and Stop processes are placed in direct competition against each other, there is a brief time window when the sensory system is already in stop mode while the motor system remains in Go mode (Chapter 6), suggesting sensory suppression processes can be switched off more rapidly than motor execution processes. Sensory suppression processes can also be switched *on* rapidly, are responsive to ongoing changes in the motor plan (Chapter 7), and can be modulated by attention (Chapter 8). Motor-sensory interactions are programmed at the level of individual movements, not sequences (Chapter 9). Finally, Chapter 10 provided evidence for a subcortical contribution to sensory suppression. When a prepared movement is triggered involuntarily by an acoustic startle stimulus, reaction time is accelerated, and sensory suppression processes are accelerated to match.

11.1 Contributions to current knowledge

11.1.1 Motor and cognitive origins of sensory suppression

In all experiments reported in this thesis, a strong motoric component of sensory suppression was evident in the form of classic premovement sensory suppression curves (see e.g. Figures 2.7 and 3.4). This confirms that sensory suppression can be represented at the level of motor execution (Williams et al. 1998; Williams and Chapman, 2000; 2002; Chapman and Beauchamp, 2006). Further evidence for a relatively sub-cognitive form of sensory suppression is the finding that attenuation occurs only for the first element in a motor sequence (Chapter 9). Even in circumstances where the reaction time is artificially accelerated by an acoustic startle, premovement sensory suppression remains intact (Chapter 10). Taken together, these findings suggest that sensory suppression is a very robust motoric phenomenon linked to movement generation.

However, sensory suppression can also have a strong cognitive component as demonstrated by other experiments in this thesis. We found evidence that preparation alone can gate sensory processing. There was an element of sensory suppression associated with actions which are prepared, but then inhibited before execution (Chapters 3-5). Thus, cognitive factors play an important role in the suppression of tactile sensations prior to movement. Furthermore, sensory suppression was also observed in the preparatory foreperiod of a trial, well in advance of muscle activation and movement onset (Chapter 4) providing further evidence for the cognitive nature of sensory suppression. Some of these contributions will now be discussed in more detail.

11.1.2 Sensory suppression and cognitive processes with no motor output

A finding of this thesis is that movement does not need to be a criterion of sensory suppression. The sensory-detection task revealed that cognitive processes which do not have a motor output can be similar to processes that do result in movement. Inhibitory

processes can be dynamic, just like go processes. They can have a measurable duration and unfold in a way that is similar to execution processes. The stopping process has traditionally proven difficult to study since stop and NoGo trials often do not produce overt behaviour. Inhibition of motor responses is a concealed operation. As a result there is no reaction time to measure. Therefore, behavioural studies often need to rely on the mathematical estimation of the internal latency of stopping (Chapters 5 and 6). However, these mathematical models (e.g. the "horse-race model") typically only determine the start and stop times of the race.

The release from sensory suppression following the NoGo signal offers a new way to measure the "stopping" process (Chapter 5). When a movement is prepared but then cancelled, the sensory system needs to be adjusted accordingly. Our results suggest that stopping is not a single discrete event, but rather is a process that develops gradually and monotonically over time. Interestingly, the sensory-detection task revealed that the rate of sensory suppression on Go trials proceeds more quickly than the rate of recovery from suppression on NoGo trials (Chapter 5). Therefore, an advantage of the sensory-detection (S-D) task is that it can be used to measure the internal structure of a process and not just the start- and end-points. The S-D task offers a new way to measure inhibitory processes and also can map events that occur during the "race" (Chapters 5 and 6). Our results demonstrate that the sensory system can be active when the motor system is not (Chapters 3-7). Similarly, Bays et al. (2005; 2006) observed that self-induced inputs are suppressed only when the subject expects to generate sensory input and that actual movement was not necessary for attenuation.

Experiments 4(a) and 4(b) raise the question as to long it takes to dismantle a prepared motor command (see Chapter 5). Experiment 4(a) showed that significant dismantling of the prepared motor command happens within the first 100 ms after the NoGo signal, although some further residual dismantling occurred later between 100 ms and 200 ms after the signal (see Figure 5.4). Experiment 4(b) demonstrated that the onset of dismantling is rapid, activating within the first 25 ms after the NoGo signal. However, the magnitude of this dismantling effect was similar to that observed in Experiment 4(a),

and yet was much faster (see Figure 5.9). Taken together these findings seem to suggest that dismantling takes place over whatever time-course the experiment measures. It should be noted that the two experimental protocols were slightly different (see Chapter 5). Therefore the results of neither experiment are comprehensive. While both show a consistent slope of dismantling, the time window for each experiment differs. Future experiments could systematically investigate the "real" duration of dismantling.

11.1.3 Motor and sensory outputs can be dissociated

Sensory output fluctuates with motor output (Williams et al., 1998; Williams and Chapman, 2000). Generally, as the motor system is inhibited the sensory system is disinhibited and there is release from sensory suppression. In this way, both the motor and the sensory systems complement each other so that each movement is accompanied by appropriate sensory processing. Interestingly, behaviour can sometimes dissociate from its sensory consequences. When Go and Stop processes are placed in maximum competition against each other, there is a brief time window when the sensory system is in Stop mode while the motor system is in Go mode (Chapter 6). These cases are probably rare but are of considerable interest as they reveal how the motor and sensory systems interact dynamically. The brief independence of sensory and motor systems suggests that the balance of excitation-inhibition can be set separately for each system.

11.1.4 Premovement sensory suppression and backward masking

In a recent study, Chapman and Beauchamp (2006) suggested an alternative "backward masking" explanation of premovement sensory suppression effects during abduction of a digit. This is important. If premovement sensory suppression is due to backward masking, then it would call into question the cognitive contribution to premovement sensory suppression made previously (Williams et al., 1998; Williams and Chapman, 2000; 2002; Walsh and Haggard, 2007). According to the backward masking

interpretation, the sensation of a first target stimulus could be attenuated by the subsequent presence of a second more powerful masking stimulus. Thus, movement of the index finger could potentially mask the perceived intensity of the weak electric shock.

Backward masking critically occurs when the mask is presented within 100 ms of the target stimulus (Laskin and Spencer, 1979). If sensory suppression was found to occur outside of the critical period, then this would provide strong evidence against a backward masking explanation for premovement attenuation. This is exactly what was demonstrated in Chapter 4 when sensory suppression was observed in the preparatory foreperiod of a trial, well in advance of muscle activation and movement onset. We observed that sensory suppression was present more than 250 ms before movement onset and in advance of a trigger signal. This finding cannot easily be explained by backward masking (Chapman and Beauchamp, 2006) and strongly suggests that pre-signal sensory suppression instead arises because of the cognitive effects of preparation. A contribution of the present thesis is therefore that sensory suppression has a strong cognitive component.

11.1.5 Premovement sensory suppression and the awareness of action

Preparatory activity takes place in the central nervous system before an action begins. The question of which components of premotor preparation are available to awareness does not currently have a clear answer (Blakemore et al., 2002). Traditionally the processes that occur prior to movement have proven very difficult to study because of their implicit nature. One of the few methods available for the experimental study of the awareness of action was developed by Benjamin Libet. Libet et al. (1983) asked subjects to look at a simulated clock with a single, swiftly rotating hand. The subjects were then asked to estimate when they first felt the "urge" of wanting to move (the so-called "W judgement") when performing spontaneous movements. On average, subjects reported awareness of the urge to move approximately 200 ms before the onset of muscle activity. This suggests that conscious intention occurs immediately prior to movement. However,

Libet et al.'s (1983) findings have been widely criticised. For instance, the validity of asking subjects to report when they feel an "urge" has been questioned (e.g. Danquah et al., 2007; Gomes, 2002). In Libet-type studies the numerical value subjects report for their W judgment can vary strongly depending on how subjects divide their attention between the clock and their internal stream of consciousness.

The sensory detection task presented in this thesis provides an alternative means of measuring the events that occur just prior to movement. In this task, subjects simply have to report whether they felt a weak electric shock or not. Though subjects may occasionally have difficulty in detecting the presence of a shock due to movement-related attenuation, they have no difficulty in understanding and engaging with the task. By analysing subjects' response patterns and the timing of the shocks a clear picture of sensory events prior to movement can be mapped out.

Overall, premovement sensory suppression and Libet's W judgement are both measures of anticipatory processes prior to movement. Yet, they may reflect different aspects of motor preparation. The timing of both events is different (however, see Klein, 2002). The W judgement usually occurs approximately 200 ms before muscle onset (e.g. Lau et al., 2004). Premovement sensory suppression begins slightly later, approximately 100 ms before the onset of EMG activity. While Libet's W judgement seems to provide a subjective measure of the anticipatory awareness of action i.e. of the feeling of about to do something, the validity of the method has been widely criticised. The method requires subjects to introspect and report the time of an "urge". In contrast, premovement sensory suppression provides a clearer, more objective method for measuring anticipatory movement-related processes.

11.1.6 Premovement sensory suppression and the sense of agency

The term 'sense of agency' describes the feeling that 'I' am a source of action that can control events in the outside world. The subjective experience of intention allows us to

recognize whether an external event was linked to our own action or not, and thus to have a sense of agency (Haggard, 2005). The mind can discriminate self-generated from external events, and can thereby present us with a distinguishable subjective experience of each.

The central nervous system is thought to contain internal models which represent aspects of one's own body and its interaction with the environment (e.g. Blakemore et al., 2002). A planner or inverse model selects motor commands appropriate for the goal. The motor command is sent to the relevant muscles and at the same time an efference copy of the motor command is sent to an internal forward model. The forward model then makes predictions of the sensory feedback based on an efference copy of the motor command. When the actual and the predicted sensory feedback are of equal magnitude they cancel, and less sensory information from the movement is processed. According to several theories, this cancellation is used to distinguish our own actions from externally-produced sensory stimuli (Jeannerod, 1988; Wolpert, 1997). By comparing the predicted to the actual sensory feedback, it is possible for the central nervous system to distinguish the sensory consequences of our movements from sensory signals due to changes in the outside world i.e. to attribute a sense of agency. The absence of feedback can therefore inform the motor system that 'I' am the source of action.

Motor control models as described above, propose that the extent to which tactile stimulation is attenuated provides the motor system with a clue as to whether the stimulation is external or due to self-movement. However, signal amplitude alone may not always be a good guide for the attribution of agency. It is not clear how the motor system can distinguish a weak externally-generated stimulus from the sensory effects of self-generated movement that have been attenuated and are of the same quality and magnitude. Furthermore, artificial externally-imposed stimuli such as electric shocks are also suppressed, suggesting that gating is general.

When an artificial delay is introduced between a movement and the resultant tactile stimulation, there is a corresponding decrease in the level of sensory attenuation and an

increase in 'tickliness' (Blakemore et al., 1999). Therefore, precise timing seems to be a key aspect of our sense of agency. The experiments in this thesis suggest that premovement sensory suppression can act rapidly and in a very time-specific way. When subjects switched from going to stopping (Chapter 6) or vice versa (Chapter 7) or were startled (Chapter 10), sensory modulation was rapid and powerful. Sensory suppression shows precise timing characteristics which closely match the timing of motor output. Explicit judgements of agency also involve precise timing of efferent signals (Tsakaris et al., 2005). The present results confirm that the timing of both sensory and motor processing may play a key role in both the experience and the judgement of agency.

11.1.7 A subcortical contribution to premovement sensory suppression

Chapter 10 investigated the effects of the startle acceleration of response on premovement sensory suppression. Startle acceleration of voluntary reactions produced a corresponding acceleration of sensory suppression. This result suggests that the subcortical structures governing fast, automatic motor output also have access to the same circuits that are involved in movement-related modulation of sensations (Walsh and Haggard, 2008). Thus, another contribution of this thesis to current knowledge is the possible identification of a new pathway from the reticular formation to the system(s) governing sensory processing (see Chapter 10).

11.2 A framework for premovement sensory suppression

In the following section a framework for sensory suppression providing an overview of some previous research is presented. The framework may be useful when considering at what level of the central nervous system sensory suppression is happening. Figure 11.1 also integrates the influential basal ganglia model of voluntary movement (see Figure 6.2; Nambu et al., 2002). It is argued that this model fits with the results of Chapters 6 and 7 and may also help guide future research.

Research has identified many *neural loci* or sources of sensory suppression signals (see Section 1.4.3). Cortical stages of the motor hierarchy which prepare motor commands can contribute to the production of sensory suppression of voluntary movements (e.g. Haggard and Whitford, 2004; Jiang et al., 1990b; Voss et al., 2006). Similarly, other studies have proposed *destinations* of sensory suppression at the level of the spinal cord (see Section 1.4.4; Seki et al., 2003), the dorsal-column nuclei (e.g. Gordon and Jukes, 1964) and S1 (e.g. Rushton et al., 1981; Chapin and Woodward, 1982). An overview of these studies and others is presented in Figure 11.1.

The brain has a number of mechanisms at its disposal that can operate at various levels within the motor hierarchy. Depending on the context, one or more of these mechanisms may be deployed to ensure motor-sensory behaviour that is appropriate to the specific situation. While many studies have been carried out on sensory suppression, they typically only examine overall input-output relations making it hard to distinguish the origins of sensory suppression from the destinations. Therefore, the origins, sites and manifestations of sensory suppression can sometimes be confused. An important next step is to try and fully describe the neuroanatomical loci of the mechanisms involved in these motor-sensory interactions i.e. to fill in the gap between the two vertical dashed lines in Figure 11.1 by joining the heads of the arrows for the "neural loci" with the tails of the arrows for the "destinations" of sensory suppression.

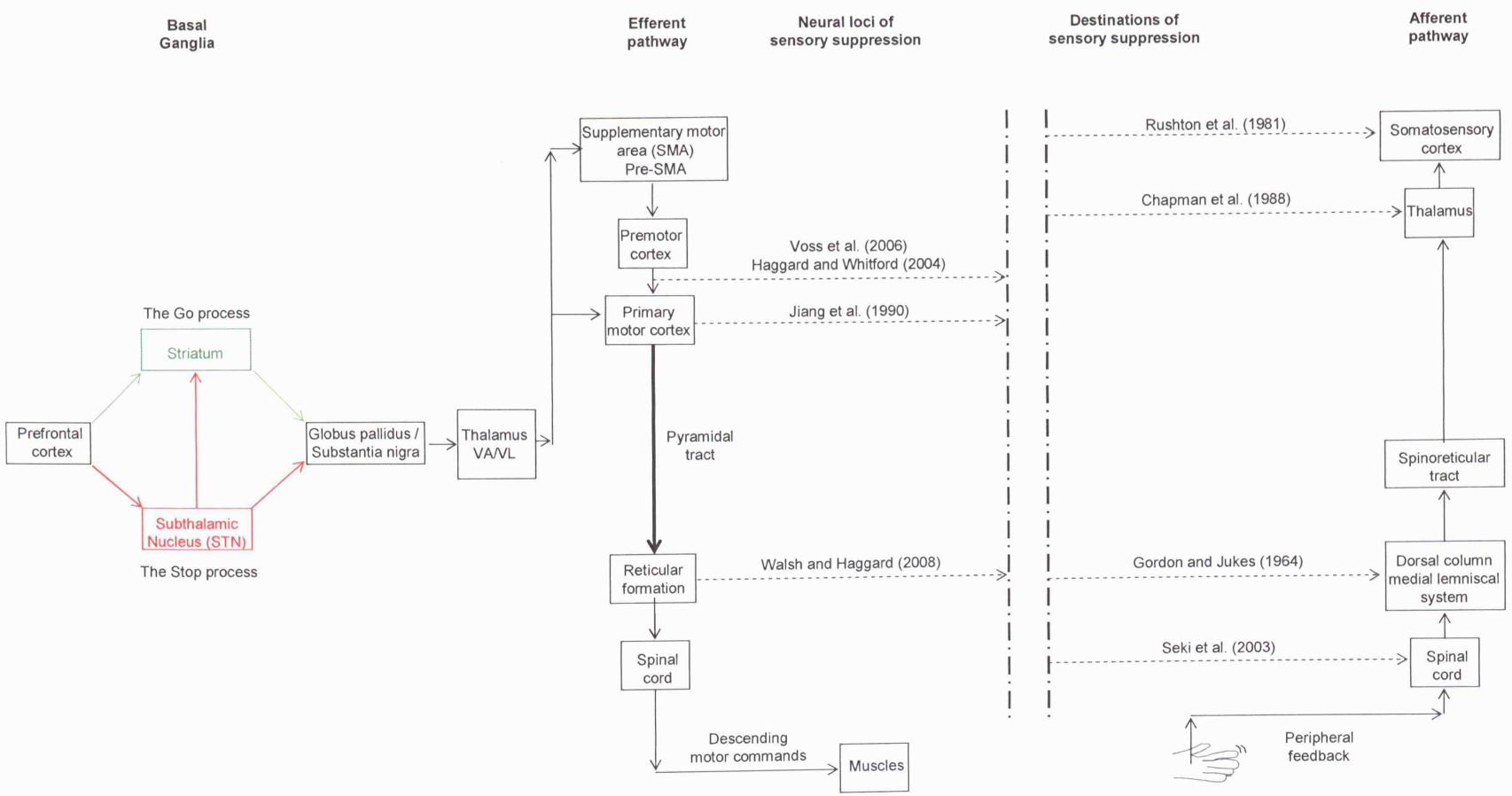

Figure 11.1. Global schematic of efferent and afferent pathways showing possible interactions for premovement sensory suppression including a new efferent "reticular formation" pathway (Walsh and Haggard, 2008; see Chapter 10). Also shown is the influential basal ganglia model (Nambu et al., 2002) which may influence premovement sensory suppression via the thalamus (see Chapters 6 and 7). An important next step for research is to try and fill in the gap between the two vertical dashed lines by joining the heads of the arrows for the "neural loci" with the tails of the arrows for the "destinations" of sensory suppression.

All of the experiments in this thesis suggest that the sensory system fluctuates as a function of the motor system. Thus, when a subject prepares to move there is a reduction in sensation in the moving limb prior to and during movement. However, when a prepared movement is cancelled, sensory suppression is released as the motor and sensory systems re-adjust. In Chapters 6 and 7, using the stop-signal paradigm (Logan, 1982), it was shown that premovement sensory suppression can be modulated. These results are clearly explained using the basal ganglia model for the control of voluntary movement (Nambu et al., 2002). Go and stop processes have been linked to different neural pathways (Goldman-Rakiç, 1987). Go and stop processes compete via "direct" and "hyperdirect" pathways through the basal-ganglia. The balance of stopping and going is thought to select action or action inhibition (Nambu et al., 2002). Going significantly activates frontal, striatal, pallidal and motor cortical regions (Aron and Poldrack, 2006). This pattern of activation is consistent with the "direct" pathway (Nambu et al., 2002). Stopping significantly activates right inferior frontal cortex (IFC) and the subthalamic nucleus (STN; Aron and Poldrack, 2006) via the "hyperdirect" pathway (Nambu et al., 2002). The speed of Go and Stop processes is thought to relate to the relative activation of these neural pathways (Aron and Poldrack, 2006).

There is general consensus that the go process excites thalamocortical output while the stop process blocks it (Alexander and Crutcher, 1990; Aron and Poldrack, 2006; Garavan et al., 1999). The nucleus reticularis thalami may be involved in modulating sensation during motor activity. This structure is a specific thalamic nucleus responsible for transmitting modulatory basal ganglia input into the afferent somatosensory system (Brunia, 1999; Töpper et al., 1993). Motor and premotor cortical areas, which are under basal ganglia control, are also able to modulate somatosensory input at prethalamic, thalamic and cortical levels (see Figure 11.1; Chapman 1994; Chapman et al., 1988; Coquery, 1978; Jiang et al., 1990; Jones, 1986; Shin and Chapin, 1990). It is known that basal ganglia disorders, such as Parkinson's (PD) and Huntington's disease (HD) present with sensory deficits. Somatosensory evoked potentials (SEPs) are impaired in cortical areas that receive modulatory somatosensory input via the basal ganglia (Töpper et al., 1993). Parkinson's and Huntington's disease are characterised by abnormal cortical and

subcortical activation during passive sensory stimulation (Boecker et al., 1999). Also, the subthalamic nucleus (STN) has been specifically implicated in Parkinson's disease (van den Wildenberg et al., 2006). It is proposed that the neural structures described in the basal ganglia model (Nambu et al., 2002) may underlie the effects observed in Chapters 6 and 7. Taken together, the model and framework may offer a useful starting point for the further investigation of sensory suppression during response execution and inhibition, and may help generate useful hypotheses for future research.

11.3 Methodological considerations

The electrical stimulation used in this study presented a reliable means of providing stimuli of identical duration and detectability. Subjects reported that the electrical stimuli did not feel unnatural. By calibrating stimulus intensity relative to a given detection level, baseline detection performance at rest was identical and comparable from subject to subject permitting the pooling of data from multiple subjects. Detection thresholds were measured both before and after the experiment and were compared statistically, thereby ensuring that there was no systematic change in shock detection rates during an experiment. However, a number of methodological considerations need to be taken into account.

11.3.1 The relationship between movement parameters and perceptual performance

An important methodological consideration is whether the presence of the shock affected preparation or execution of the ensuing movement. There may have been a differential trade-off between motor preparation and shock detection as subjects allocated limited attentional resources to the detection of the shock at the expense of preparation for movement. Therefore, this may account for the presence of time-varying detection rates for the right shock stimulus hand and the absence of time-varying detection rates for the left hand where no shock stimulus was applied (Chapter 2). It is known that movement-

related gating is a function of the kinematics of the movement, with faster movements producing larger gating effects (Angel and Malenka, 1982).

Previously, Williams et al. (1998) tested if the time-dependent decrease for the right (shocked) hand were due to modifications in perceptual performance related to variations in kinematic parameters including peak amplitude, peak velocity and peak acceleration. Data were divided into two groups: trials in which the shock stimulus was applied before the onset of EMG and trials in which the shock stimulus was applied after the onset of EMG. For each group, the kinematic parameters were compared across trials in which the shock stimulus was, or was not perceived. No significant differences were found. Ideally, the current design would have included checks to verify that there was no difference in kinematic parameters between the left hand and the right shock-stimulus hand. However, EMG profiles and potentiometer outputs for the left and right hands for each subject were visually inspected and no obvious differences were evident. Furthermore, the analysis previously performed by Williams et al. (1998) suggests that when a weak shock stimulus is delivered before the movement that neither motor preparation nor execution is influenced by the presence of the shock.

11.3.2 Response bias

An advantage of the movement performed in this study, abduction of the index finger, is that it has only one major agonist (the FDI muscle). Other intrinsic and extrinsic hand muscles are likely to show small amounts of co-contraction; however these effects are likely to be minimal as the movement amplitude was limited to 20°. Subjects appeared able to detect signals at approximately the target rate of 90% at rest while minimising false positives. The rate of false positives was always extremely low throughout the series of experiments, a good indication that positive bias was not a significant factor in the experimental results. This indicates that subjects used a very conservative response strategy and subjects appeared to perform near optimally. However, in the current experimental design shock stimuli were delivered on the majority (90%) of movement

and non-movement trials. In such a design, subjects may exhibit a response bias i.e. a willingness to report the presence of a shock stimulus, which might systematically change in relation to the motor task (e.g. movement vs. non-movement). In order to control for such a bias, Chapman and Beauchamp (2006) included equal numbers of trials with and without a shock stimulus. This allowed the evaluation of the perceptual data from individual subjects using a more bias-free method based on signal detection theory (SDT; Green and Swets, 1988) and the index of detectability (d'). Chapman and Beauchamp (2006) found that the SDT method yielded virtually identical results to the method used here suggesting that changes in bias did not contribute to the current results.

A major disadvantage of the SDT method is that only 50% of trials contain a shock stimulus. Many more trials are required in order to measure a sensory suppression effect of a given size. The SDT method therefore results in experiments that are considerably longer increasing the possibility of fatigue effects. Thus, the current design attempted to obtain bias-free results while avoiding longer experiments by estimating bias from false positive detections on catch trials. This compromise in the design appears to have been successful. Fatigue effects did not appear to play a significant role in perceptual performance during the current experiments, as confirmed by the staircase procedures. Furthermore, our findings are in line with previous results (Williams et al., 1998; Williams and Chapman, 2000; 2002; Chapman and Beauchamp, 2006).

11.4 Functional significance of premovement sensory suppression

Our sensory systems are constantly bombarded by a multitude of sensory stimuli, from which we must extract the few salient stimuli that correspond to important changes in our environment. The sensory consequences that arise from our own actions potentially increase the amount of afferent information in the brain. In most circumstances, this "reafferent" information is relatively unimportant because it can already be predicted from the motor command and can therefore be discarded. Computational motor control theories (e.g. Blakemore et al., 1998; Wolpert, 1997) propose that by using a forward

model, the central nervous system can mimic sensory feedback. In this way, the outcome of an action can be estimated and used before actual sensory feedback becomes available. Thus, prediction-based modulation can act as a filter for incoming sensory signals. If the sensory consequences of a movement are predicted accurately, they are suppressed so that other more salient stimuli may be processed preferentially. According to these theories, the functional role of the movement-related suppression of perception is therefore to reduce the flow of afferent information that can be predicted from the motor command so that detection of unexpected or novel stimuli is enhanced (Bays et al., 2005; 2006; Blakemore et al., 1999; Shergill et al., 2003).

Furthermore, the premovement sensory suppression that underlies increases in perceptual thresholds prior to movement may have important functional consequences for movement preparation. During normal voluntary movements, reafferent feedback from the periphery continuously converges on spinal circuits that are activated by the descending motor commands. This input may either be synergistically combined with the motor commands or be appropriately suppressed to minimise interference. The fact that afferent input is inhibited prior to movement indicates that the brain reduces self-induced input at the earliest possible stage. A function of premovement sensory suppression might therefore be to dominate peripheral inputs that might otherwise interfere with the descending commands. The central nervous system can thereby maintain greater control of spinal circuits during voluntary movement.

11.5 Concluding comments

While at rest, we can readily feel the light touch of a feather on the back of our hand, even though only a few low threshold mechanoreceptors are activated. In contrast, when we make a voluntary movement with the same hand, the movement is not accompanied by a strong sensation of movement, even though a relatively large number of low threshold mechanoreceptor units of various types that supply the hand are activated. This implies that the inhibition of afferent inputs concomitant with movement is quite considerable. Contemporary models of motor control suggest that such afference is "cancelled" (e.g. Wolpert, 1997) or "gated" (e.g. Williams et al., 1998), i.e. that it is somehow prevented from reaching consciousness where it would serve no useful purpose. However, this does not really explain the phenomenon that is sensory suppression. Here it is proposed that the extent to which ongoing motor and sensory processes interact can give us a valuable insight into the mechanisms of voluntary control and perception.

This thesis offers three important new findings on sensory suppression which will inform future research. First, it was shown that sensory suppression occurred for actions which were prepared, but then inhibited before execution. Second, the recovery from sensory suppression offers a new method for measuring the processes triggered by a NoGo or a stop signal. Third, this thesis provided evidence for a new subcortical contribution to sensory suppression. It is concluded that sensory suppression is a useful tool for exploring the cognitive precursors of movement.

Finally, taking all into account, sensory suppression appears to be a highly general principle of organisation of motor to sensory interactions, rather than a single specific physiological circuit. Sensory suppression is not unitary. There are multiple possible sources of sensory suppression. Equally, there are multiple possible destinations of sensory suppression in the afferent somatosensory pathway. Multiple structures probably contribute to movement-related gating, and the relative contribution of each appears to vary across the chain of events that ultimately leads to perception.

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